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Management of Tobacco Budworm, Heliothis Virescens (F.) and Bollworm, Helicoverpa Zea (Boddie) in Cotton: Host Plant Resistance and Ovicidal Approaches.

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MANAGEMENT OF TOBACCO BUDWORM, *HELIOTHIS VIRESCENS* (F.) AND BOLLWORM, *HELICOVERPA ZEA* (BODDIE) IN COTTON: HOST PLANT RESISTANCE AND OVICIDAL APPROACHES

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FOREWORD

This dissertation is compiled into chapters representing a series of manuscripts ready to submit, or already submitted for publication. The Introduction and Literature Review hopefully provide a justification of these projects with an understanding of previous research in the areas of interest. All chapters are written in the style of the Journal of Economic Entomology which is published by the Entomological Society of America. The information included in this dissertation is protected under the copyrights of the journal in which the information will be published and therefore any attempt to reproduce all or part of this manuscript would violate those rights.

TABLE OF CONTENTS

TABLE OF CONTENTS (CONTINUED)

TABLE OF CONTENTS (CONTINUED)

LIST OF TABLES

LIST OF TABLES (CONTINUED)

LIST OF TABLES (CONTINUED)

LIST OF FIGURES

ABSTRACT

Growth, development and survival of pyrethroid-resistant (PY-R) and -susceptible (PY-S and FIELD-88) tobacco budworm, Heliothis virescens (F.), and bollworm (CORN-BW), Helicoverpa zea (Boddie), were compared on four cotton lines ('Deltapine 41', La. HG-660, La. HG-063 and PD-0804) and artificial diet. The FIELD-88 and CORN-BW strains had significantly higher 5 and 9 day larval weights compared to the PY-R and PY-S laboratory strains. Larvae fed squares of La. HG-660 and La. HG-063 had significantly lower larval and pupal weights, required significantly more days to pupate and had higher cumulative mortality compared to those reared on squares of a commercial cultivar, Deltapine 41.

In a field trial conducted during 1988 to evaluate advanced cotton lines and insecticide treatments against tobacco budworm and bollworm, no significant interaction was observed. However, La. HG-660 reduced larval infestations and damage, matured earlier, and had comparable yields to DPL 41. Lint turnout and boll weight was lower for La. HG-660 than that for Deltapine 41. No significant differences among treatments were found in fiber length, micronaire and fiber strength of cotton samples.

The toxicity of insecticides to eggs of PY-R, PY-S and field (FIELD-89) tobacco budworm strains was determined in laboratory tests. LC_{50}'s for all insecticides except profenofos on eggs of the PY-R strain were significantly higher than LC_{50}'s for the same insecticide on eggs of the PY-S strain. All insecticides except profenofos and methomyl were significantly more toxic to eggs of the PY-S strain compared to their respective toxicity to eggs of the FIELD-89 strain. Eggs of the PY-R strain exhibited resistance to esfenvalerate, lambda-cyhalothrin, and chlordimeform while eggs of the FIELD-89 strain possessed resistance to lambda-cyhalothrin, chlordimeform and SN 49844.

In field tests conducted during 1987-1989, all insecticide treatments except methomyl (0.071 kg [AI]/ha) and profenofos (0.56 kg [AI]/ha) exhibited initial ovicidal activity in one or more trials. The formamidines (amitraz and SN 49844) and a carbamate (thiodicarb) at 0.28 kg [AI]/ha generally exhibited residual ovicidal activity comparable to that of chlordimeform at 0.28 kg [AI]/ha.

INTRODUCTION

The tobacco budworm, Heliothis virescens (F.) and bollworm, Helicoverpa zea (Boddie), are two of the major pests attacking cotton in the mid-South (Brazzel et al. 1953, King & Phillips 1989). Historically, due to the effectiveness of insecticides, management of these pest species has relied almost entirely on chemical control. However, the ability of these insects, particularly the tobacco budworm, to become resistant to insecticides seriously hinders this strategy. In the past, as these pests have developed resistance to recommended insecticides, cotton producers have simply switched to a different class of insecticides (Sparks 1981). There are no commercial insecticides available at the present time and no experimental compounds near registration that possess a level of efficacy comparable to that of the pyrethroids.

The tobacco budworm has developed varying levels of resistance to many of the organochlorine, organophosphate, carbamate and most recently, pyrethroid insecticides used for its control in cotton (Sparks 1981, Allen et al. 1987, Plapp 1987, Luttrell et al. 1987, Leonard et al. 1988a; 1988b, Campanhola & Plapp 1989). The recent development of pyrethroid resistance in field strains of tobacco budworm has resulted in the adoption of resistance management plans (Anonymous 1986, Luttrell 1987, Plapp 1987, Roush & Luttrell 1987, Graves et al. 1988) to delay or prevent widespread

escalation of this problem. These programs may prove successful as a short-term approach, but it is unlikely they will be successful as a long-term solution to the pyrethroid resistance problem in the tobacco budworm.

Resistance management plans have recommended binary mixtures of insecticides as an alternative to using the pyrethroids as the primary chemical control against tobacco budworm and bollworm in cotton (Anonymous 1986, Plapp 1987, Graves et al. 1988). The criteria for selecting specific compounds are based on the range and density of the pest spectrum and the use of non-related insecticides from different classes with dissimilar modes of action. In addition to the synergistic toxicity of insecticide mixtures used against tobacco budworm, at least one component of the combination generally expresses ovicidal activity.

Chlordimeform and methomyl were, until recently, the only insecticides recommended as ovicides against tobacco budworm and bollworm. The importance of ovicides in resistance management strategies utilized against these pests is recognized among the pesticide producing industries and much effort is being put forth to label insecticides for their ovicidal activity. Chlordimeform, through its wide acceptance among cotton producers, has been the standard for measuring ovicidal toxicity and appeared to provide one option to be used in combination with the pyrethroids to

delay the spread of resistance and manage the tobacco budworm. However, its registration for use in cotton was cancelled by the Environmental Protection Agency in September of 1989, and it will no longer be available. Other insecticides have been evaluated for ovicidal activity against tobacco budworm and bollworm (Pitts & Pieters 1980, Horowitz et al. 1987, Bagwell & Plapp 1988), but additional information is necessary to determine the potential of chlordimeform alternatives as ovicides.

Chemical control is likely to remain a viable part of the management program for tobacco budworm and bollworm in the near future. Given the problems associated with chemical exposure in the environment, alternative long-term management strategies are necessary to maintain profitable cotton production. There are, at the present time, no commercially available alternative strategies that can cost effectively replace chemical control of tobacco budworm and bollworm. Therefore, alternative tactics must be compatible with chemical control to develop a stable overall program of insect management in cotton. One such tactic involves the use of cotton cultivars with substantial levels of resistance to tobacco budworm and bollworm and/or other cotton pests.

Plant resistance in cotton has not been utilized much in cotton insect pest management because of the difficulty

in developing cotton breeding lines with agronomically desirable qualities comparable to commercial cultivars grown under standard insect pest management systems. However, recently, the cooperative efforts of scientists from different disciplines have been successful in incorporating morphological and biochemical traits that confer resistance to tobacco budworm and bollworm into advanced cotton lines. Evidence to support these accomplishments is provided by the release of at least one commercial variety, 'DES 119', that expresses partial resistance to tobacco budworm and bollworm. There are other breeding lines with different sources of resistance to cotton pests that are showing promising results (McCarty 1987, Stringer 1987, Jones et al. 1989). Additional research with breeding lines under varying levels of cotton pest control may provide the basis for integrating plant resistance in cotton with chemical control.

As recommended insect management strategies become inadequate to cope with damaging infestations of insecticide-resistant tobacco budworm, research to provide alternative means of control is imperative to maintain stable cotton production in the future. The potential to deleteriously change the resistance situation, impact on the environment, economics and the possibility of secondary pest induction must be evaluated before such management tactics

can be incorporated into current cotton production systems. In addition, the uncertain registration status of various insecticides used on cotton further emphasizes the need for additional research to manage the tobacco budworm. This dissertation contains objectives to study these problems and aid in the development of strategies to manage tobacco budworm and bollworm, especially pyrethroid-resistant tobacco budworm.

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REVIEW OF THE LITERATURE

Development of Pyrethroid Resistance in Tobacco Budworm

Pyrethroids have been significantly more active against the tobacco budworm than the organophosphorus and carbamate insecticides used for its control in cotton (Davis et al. 1975, 1977; Harding et al. 1977). When the recommended organophosphorus insecticides failed to provide adequate field control of tobacco budworms in the mid-1970's, the pyrethroids (i.e., permethrin, fenvalerate and later cypermethrin) replaced these compounds and became extensively used. Pyrethroids comprise the basis for all strategies involved in controlling the major pests of cotton and until recently offered the most cost effective means of controlling the tobacco budworm.

Reports of field control failures involving pyrethroids first began to appear in Texas in 1985 and were later verified by laboratory tests to be due to resistance (Plapp & Campanhola 1986, Allen et al. 1987, Leonard et al. 1988a, Campanhola & Plapp 1989a). Subsequent bioassays found pyrethroid-resistant strains of tobacco budworm to express resistance in the egg, larval and adult stages to a variety of pyrethroids (Leonard et al. 1988b, Treacy et al. 1988, Campanhola & Plapp 1989b; 1989c). During 1986, the number of reported field problems that were indicated to be due to resistance became more widespread and included the states

of Arkansas, Louisiana and Mississippi (Luttrell et al. 1987, Leonard et al. 1988a). Based on the development of resistance in localized populations of tobacco budworm in several cotton growing regions, researchers in Texas and the Mid-South developed plans for resistance management to delay the occurrence of widespread resistance in the tobacco budworm and prolong the usefulness of the pyrethroids (Anonymous 1986, Luttrell 1987, Plapp 1987, Roush & Luttrell 1987, Graves et al. 1988). Additionally, a standardized monitoring procedure (adult vial test) was developed to evaluate the extent of the resistance problem and obtain a greater understanding of pyrethroid resistance in the tobacco budworm (Plapp et al. 1987, Campanhola & Plapp 1989a). Tobacco budworm infestations were less severe during 1987, 1988 and 1989 than those observed in 1985 and 1986. Fewer field control failures were reported during 1987-1989, and data from several monitoring programs indicate that resistance levels are increasing but not drastically (Graves et al. 1988, 1989, 1990; Luttrell et al. 1988; Riley 1988; Rogers et al. 1990).

Pyrethroid resistance in the tobacco budworm is apparently the result of multiple resistance mechanisms associated with metabolism, nerve insensitivity (kdr), penetration and behavior. Several studies have indicated that increased metabolism in the tobacco budworm is an

important factor in the development of pyrethroid resistance (Nicholson & Miller 1985, Sawicki 1985, Dowd et al. 1987, Little et al. 1989, McCaffery et al. 1989). Increased enzyme activity has been determined to be the primary cause of tobacco budworm resistance to the organophosphates, and this may account for the association of elevated dosage/mortality responses between organophosphates and pyrethroids in some strains of tobacco budworm. It is just as likely that resistance to the pyrethroids is related to previously used organochlorine insecticides such as DDT.

Probable mechanisms responsible for resistance to DDT (especially kdr) in the tobacco budworm have been demonstrated to be similar to those that also confer resistance to the pyrethroids (Nicholson & Miller 1985, Sawicki 1985). Dowd et al. (1987) characterized increased levels of detoxifying enzymes in a pyrethroid-resistant strain of tobacco budworm that also exhibited resistance patterns consistent with the presence of a kdr-type mechanism. McCaffery et al. (1989) observed a decrease in the amount of pyrethroid penetrating and reaching the target site in strains of pyrethroid-resistant tobacco budworm as compared to a susceptible strain. Furthermore, Sparks et al. (1988) demonstrated an increase in kdr and a decrease in movement (capable of influencing uptake) in a strain highly resistant to the pyrethroids. Thus, the current data

indicate that multiple factors are responsible for pyrethroid resistance in the tobacco budworm.

Advances in Developing Cotton Breeding Lines Expressing Resistance to Tobacco Budworm and Bollworm

Over the past several decades, significant advances in the understanding of tobacco budworm and bollworm interactions with cotton plants have been made. This information has enabled researchers to identify, isolate and transfer biochemical and morphological traits conferring plant resistance in cotton to tobacco budworm and bollworm in cotton. Recent research has targeted the direct effects of resistant traits in cotton on the physiology of tobacco budworm and bollworm and the indirect effects of these traits on insecticide susceptibility of these pests. This review briefly summarizes the research findings in the aforementioned areas that are pertinent to the objectives in this dissertation.

Allelochemicals

Cotton plants have an abundant supply of secondary products that are capable of influencing insect development and survival. These compounds vary qualitatively and quantitatively during plant growth and in different plant structures. The selective feeding nature of tobacco budworm

and bollworm coupled with their high metabolic capacity very often enables these pests to survive in the presence of many of the allelochemicals found in cotton.

Probably more work has been done on the terpenes (primarily gossypol) than any other group of secondary plant compounds in cotton. Terpenes, primarily gossypol, are the most abundant allomones found in cotton and are associated with pigment glands located on leaves, stems and reproductive structures. Gossypol is not the only terpene found in cotton; however, it is found in higher amounts and than others that have currently been identified (Bell & Stipanovic 1977, Hedin et al. 1981; 1983). Several of the other terpenes, heliocides H_1-H_4 and hemigossypol, are noteworthy because of their toxicity to tobacco budworm and bollworm (Elliger et al. 1978, Hedin et al. 1983). Sappenfield et al. (1974) used the relationship of pigment glands and terpene content in the flower bud to establish a rating system for the evaluation of breeding lines suspected of containing this trait.

Numerous laboratory and field studies have substantiated high gossypol as conferring resistance to tobacco budworm and bollworm. Larvae subjected to gossypol and other terpenes incorporated into artificial rearing media have expressed significantly lower survival, reduced growth rates and require longer to pupate and emerge as

adults (Bottger & Patana 1966, Lukefahr et al. 1966, Shaver & Parrot 1970, Shaver et al. 1978, Hedin et al. 1983). Similar results have been produced in forced feeding studies using flower bud or leaf tissue with high terpene content (Mullins & Pieters 1982, Mulrooney et al. 1985, Parrott et al. 1983). In field tests, breeding lines with high glandulosity on flower buds have significantly reduced tobacco budworm and bollworm larval infestations and damage (Lukefahr & Houghtaling 1969, Zummo et al. 1983, Stringer 1987, Jones et al. 1989). In several instances, under high tobacco budworm and bollworm pressure, these lines have produced yields comparable to or greater than commercial varieties in the absence of chemical control (Jones et al. 1989).

There are other allelochemicals isolated from cotton plants shown to influence feeding and development of tobacco budworm, bollworm and other cotton insect pests (Bell 1986). Several flavonoids including condensed tannins, anthocyanidins and flavonols have demonstrated antibiotic activity against tobacco budworm and bollworm (Shaver & Lukefahr 1969, Chan et al. 1978, Schuster & Lane 1979, Hedin et al. 1981; 1983, Mulrooney et al. 1985).

Other allelochemicals not yet identified in cotton plants have been qualitatively demonstrated to reduce tobacco budworm and bollworm development and survival in

laboratory tests and to reduce damage ratings relative to commercial cultivars in field trials. Lukefahr et al. (1974) found unknown biochemical mechanisms ('X' factors) in cotton racestocks from Mexico (T-254 and T-27) that negatively influenced tobacco budworm and bollworm development in the laboratory and decreased damage ratings in field tests. Subsequent bioassays have classified the 'X factor' in T-254 as being a mixture of allelochemicals including condensed tannins and terpenoids (Stipanovic et al. 1976, Chan et al. 1978). Another unidentified allelochemical has been termed the 'Q' factor and is found in the South Carolina PD breeding lines (Culp et al. 1979). These lines express variable levels of resistance to tobacco budworm and bollworm in field tests. The results are, in part, influenced by high amounts of rainfall and suggest this allelochemical has water soluble (Bhardwaj et al. 1985).

Morphological Traits

Variations in cotton plant morphology offer an additional source of resistance to manage the tobacco budworm and bollworm complex in cotton. Adults prefer to oviposit on leaves of pubescent cotton. Increasing the density of pubescence on leaves has been shown to inhibit movement of early stage larvae to flower buds thus increasing the time they are exposed to predators and

parasitoids (Ramalho et al. 1984). The glabrous or smooth leaf trait confers antixenosis to tobacco budworm and bollworm by providing a non-preferred site for oviposition. Several studies have verified a reduction in the number of eggs on smooth leaf cotton compared to cottons with normal pubescence (Lukefahr et al. 1965, Lukefahr et al. 1971, Robinson et al. 1980). Reduced pubescence also can enhance the effectiveness of predators and parasitoids by increasing the searching efficiency.

Large glands (nectaries) on the cotton plant located on the underside of leaves, at the base of the bracts subtending the flower bud and on the floral structures provide an important source of food and water for many adult insects common in cotton fields. The removal of nectaries from cottons lines has been associated with a reduction in tobacco budworm and bollworm oviposition (Lukefahr et al. 1965, Meredith et al. 1973, Schuster & Maxwell 1974). This trait is only effective in suppressing oviposition in large fields planted to nectariless cotton and not in small plot trials because of interplot migration of adults. Removal of nectaries also has a negative impact on hemipteran and neuropteran predators by reducing a source of food for adults (Schuster & Maxwell 1974, Schuster et al. 1976).

Modifying leaf and bract shapes has not been shown to directly influence tobacco budworm and bollworm

infestations. The open bract (frego-bract) trait and okra leaf shape have a more open leaf canopy compared to normal leaf cottons that significantly improves insecticide penetration within the plant and on the flower bud (Parrot et al. 1973, James & Jones 1985). Although improvements in distribution of insecticides within the plant have not significantly increased tobacco budworm and bollworm control, the potential to use the minimum effective rate of insecticides to achieve satisfactory control may be increased.

One final morphological trait that may add a supplemental source of resistance to tobacco budworm and bollworm is associated with pollen color. Several studies have shown that growth and development of larvae are negatively affected by diets containing yellow or orange pollen compared to larvae fed white or cream-colored pollen (Hanny et al. 1979, Bailey 1981). This mechanism of resistance is probably through antibiosis and is related to variations in content of allelochemicals among the different colored pollens (Hanny et al. 1979).

Cotton Plant Resistance and Insecticide Toxicity Interactions

Several laboratory and field studies have shown that insecticidal toxicity to tobacco budworm and bollworm can be modified by altering the morphological traits of the

cotton plant or by adding cotton allelochemicals to their larval diets. Changing the profile of the bracts or leaves from normal shapes to more exposed flower buds and open canopied foliage has been shown to slightly improve application efficiency and has the potential to increase control of tobacco budworm and bollworm in cotton (Lincoln et al. 1971, Schuster & Anderson 1976, James & Jones 1985). Increasing the amount of gossypol in artificial diet has been demonstrated to significantly decrease the toxicity of methyl parathion to tobacco budworm and bollworm larvae (Perkins & Canerday 1971, Shaver & Wolfenbarger 1976, Muehleisen et al. 1989). However, if tobacco budworm larvae are predisposed to cotton flower buds containing high concentrations of gossypol, they are more susceptible to insecticides (Mullins 1980, Muehleisen et al. 1989). These differences between rearing on host and diet may be associated with other allelochemicals in cotton flower buds interfering with gossypol. Also, artificial diets are usually super optimal for larval growth and development which may partially compensate for the detrimental effects of insecticides. Although unclear, the relationship between allelochemical content in host plants and insecticidal toxicity to tobacco budworm and bollworm should be further explored.

Xenobiotics as plant allelochemicals at sublethal doses

are generally thought to be metabolized in the same manner as insecticides, i.e. via detoxification enzyme systems in most insect species including tobacco budworm and bollworm (Dowd et al. 1983, Terriere 1984, Mullin 1985, Muehleisen et al. 1989). Furthermore, the activity of these enzyme systems may be induced or increased in tobacco budworm larvae by exposure to allelochemicals in diets (Brattsten 1987b). Induction of enzymes is more effective in increasing metabolic defenses in insects that already possess high activities (Terriere 1984). An insecticide-susceptible tobacco budworm strain that has high insecticide metabolizing enzyme activities can be selected with plant allelochemicals and further increase those enzyme activities (Brattsten 1987a, 1987b). Brattsten (1987b) found an increase in cytochrome P-450 activity, N-demethylation, epoxidation and glutathione transferase activity in tobacco budworm could be induced by several cotton allelochemicals. However, gossypol, one of the more important compounds in cotton, did not influence enzyme activity in tobacco budworm. Because pyrethroid resistance in tobacco budworm has been characterized by increases in mixed function oxidase and esterase enzyme activity, there is the potential for cotton allelochemicals to be less active against insecticide-resistant tobacco budworm, particularly if the enzyme systems involved in detoxification of both

xenobiotics are similar. This relationship becomes extremely important if breeding lines with tolerance to tobacco budworm and bollworm are combined in management strategies of pyrethroid-resistant tobacco budworm.

Ovicidal Toxicity of Selected Insecticides to Tobacco Budworm and Bollworm

Noctuid eggs are very susceptible to ovicides immediately after oviposition before the membranes surrounding the embryo are fully formed (Salkeld & Potter 1953, Smith & Salkeld 1966). Previous studies (Wolfenbarger et al. 1974, Chalfant et al. 1979, Pitts & Pieters 1980) have attributed most of the egg mortality from insecticides as being due to direct application. However, eggs oviposited after treatment may be subjected to insecticide exposure by residues on leaf surfaces and/or by insecticide vapor (fumigant). Toxicity to insect eggs from fumigant activity of selected insecticides (particularly formamidines) has been demonstrated previously (Smith & Salkeld 1966, Phillips 1971, Wolfenbarger et al. 1974).

Larval mortality immediately following eclosion from the egg may be caused by several factors that are associated with insecticide exposure during the egg stage. These include chorion feeding, contact and/or oral toxicity from

insecticide residues on the piece of cotton foliage upon which the egg was oviposited, or a combination of both. Gonzales and Allen (1985) and Bradley and Agnello (1988) observed considerable mortality wherein the larvae died partially eclosed from egg directly and indirectly exposed to insecticides. They concluded this mortality was due to feeding on the chorion that had been exposed to sublethal doses of the insecticide that were incapable of halting embryo development. In many cases it has been difficult to interpret tobacco budworm and bollworm mortality resulting solely from the ovicidal activity of insecticides.

Because most tobacco budworm and bollworm eggs are oviposited on exposed surfaces of the cotton plant (Brazzel et al. 1953, Farrar & Bradley 1985), they are generally susceptible to insecticides possessing ovicidal properties. Until recently, only two compounds, chlordimeform and methomyl, have been used as ovicides to control the tobacco budworm and bollworm complex on cotton (Pitts & Pieters 1980). Other compounds demonstrating some degree of ovicidal activity against tobacco budworm and bollworm include amitraz (Coulon 1978, Pitts & Pieters 1980, Bagwell & Plapp 1987, Horowitz et al. 1987), fenoxycarb (Masner et al. 1987), fenvalerate (Horowitz et al. 1987), flucythrinate (Jany 1984), permethrin (Tysowsky & Gallo 1977), profenofos (Campbell et al. 1979), sulprofos (Herzog & Phillips 1987)

and thiodicarb (Gonzales & Allen 1985, Bradley & Agnello 1988). In a detailed laboratory study, Horowitz et al. (1987) reported differences in toxicity of several pyrethroids, carbamates and organophosphates to tobacco budworm eggs. This experiment also demonstrated varying levels of tolerance to the same insecticides when tested against field collections of tobacco budworm eggs.

Bradley and Agnello (1988) also reported significant changes in the toxicity of several insecticides in samples of tobacco budworm and bollworm eggs collected at several intervals posttreatment. They indicated a saturation point is reached with some insecticides at which an increase in the rate did not result in an increase in the toxicity to eggs. Campbell et al. (1979) reported the optimum rate of chlordimeform to be 0.281 kg [AI]/ha, and control could not be improved by increasing the rate above this level. These results also indicated that rates of chlordimeform lower than 0.280 kg [AI]/ha did not provide acceptable control of tobacco budworm eggs. Therefore, it appears that for chlordimeform and possibly for other insecticides that possess ovicidal activity, the optimum range of activity is somewhat narrow.

Ovicides used alone are generally not capable of economically managing tobacco budworm and bollworm in cotton. Thus, the most common use would be as a component

of a binary mixture with an effective larvicide. Horowitz et al. (1987) examined such insecticide combinations against tobacco budworm eggs and reported synergistic properties of ovicides in combination with pyrethroids. Such mixtures have already been recommended in the mid-South pyrethroid resistance management strategies for delaying widespread resistance development (Anonymous 1986, Luttrell 1987, Graves et al. 1988). Insecticide resistance to available larvicides in the tobacco budworm increase the need for discovery and development of ovicides.

The preceding review of available literature has served to introduce the current problems and progress associated with management of tobacco budworm and bollworm in cotton. The following objectives were formulated to provide information on specific areas of research to improve integrated pest management of these species in cotton.

List of Research Objectives

I. To evaluate cotton plant resistance against tobacco budworm and bollworm and determine its potential as a component of a management system for these pests.

   A. Determine the influence of selected cottons on growth, development and survival of pyrethroid-resistant and -susceptible tobacco budworm and bollworm

   B. Compare tobacco budworm and bollworm resistant cotton breeding lines under reduced insecticide use strategies to the currently recommended tobacco budworm and bollworm control tactic.

II. To determine the efficacy of selected insecticides against tobacco budworm and bollworm eggs.

   A. Determine the relative toxicity of selected insecticides to pyrethroid-susceptible and -resistant tobacco budworm eggs in laboratory tests.

   B. Compare the ovicidal efficacy of selected insecticides in field screening trials against tobacco budworm and bollworm.

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CHAPTER I

GROWTH, DEVELOPMENT AND SURVIVAL OF PYRETHROID-RESISTANT AND -SUSCEPTIBLE TOBACCO BUDWORM AND BOLLWORM (LEPIDOPTERA: NOCTUIDAE) ON SELECTED COTTONS

Introduction

The tobacco budworm, Heliothis virescens (F.), and bollworm, Helicoverpa zea (Boddie), continue to persist as cotton, Gossypium hirsutum L., pests of major importance in the United States (King & Phillips 1989). In an integrated system of management, insecticides have been the primary tool for controlling these insects. The recent development of pyrethroid resistance in field populations of tobacco budworm has made the future role of these insecticides in cotton insect pest management uncertain. Research and extension programs have been directed towards the reduction of chemical applications for the control of tobacco budworm and bollworm in cotton (Herzog et al. 1988), but the loss of the pyrethroids will cause researchers to intensify the search for alternative management tactics. Under the pest management programs currently employed in cotton, plant resistance has demonstrated considerable potential as an additional tool to control the tobacco budworm and bollworm.

For plant resistance in cotton to be successful, the mechanisms of resistance should be effective against these pests regardless of their insecticide susceptibility. Insects generally metabolize insecticides and plant toxins in a similar manner, and the enhanced capacity to detoxify a specific xenobiotic may cause an increase in tolerance to

other xenobiotics (Dowd et al. 1983). Several studies have shown that insect sensitivity to insecticides is influenced by the host plant on which they fed (Markos & Campbell 1943, Wieb & Radcliffe 1973, Kea et al. 1978, Combs & Chambers 1979, El Refai et al. 1979, Berry et al. 1980, Wood et al. 1981, Yu 1983, Hinks & Spurr 1989). Gossypol, an important allelochemical in cotton plants, has been shown to be antagonistic to the toxicity of insecticides tested against the tobacco budworm, bollworm and Spodoptera littoralis (Boisduval) (Perkins & Canerday 1971, Abou-Donia et al. 1974, Shaver & Wolfenbarger 1976). Furthermore, plant toxins are capable of directly influencing the activity of enzymes which are important in insecticide detoxification (Brattsten et al. 1977, Brattsten 1983, Dowd et al. 1983, Yu 1983, Brattsten 1987b). No previous reports have related insecticide resistance in insects to decreased susceptibility to allelochemicals in host plants.

Insects that have high levels of detoxifying enzyme activity and possess the capacity to develop metabolic insecticide resistance may better tolerate allelochemicals in resistant host plants. The tobacco budworm has developed resistance to a variety of insecticides (Sparks 1981, Leonard et al. 1988) which may be, in part, related to relatively high levels of insecticide detoxifying enzyme activity (Brattsten 1987a, 1987b). Pyrethroid resistance

in tobacco budworm is associated with increased metabolic activity through mixed function oxidase and esterase enzyme systems (Dowd et al. 1987, Little et al. 1989, McCaffery et al. 1989). This may influence the ability of pyrethroid-resistant tobacco budworms to metabolize secondary plant compounds. It is the purpose of this study to evaluate the growth, development and survival of pyrethroid-resistant and -susceptible strains of tobacco budworm and bollworm on selected cotton lines and artificial diet.

Methods and Materials

Tobacco Budworm and Bollworm Strains

One bollworm (CORN-BW) and three tobacco budworm (PY-S, PY-R, and FIELD-88) strains were used in this study. The bollworm strain was initiated from three collections of larvae taken from fields of sweet corn, Zea mays L. These samples consisted of ca. 80 larvae collected on 16 June 1988 (Northeast Research Station, St. Joseph, La.), 175 larvae collected on 20 June 1988 (Red River Research Station, Bossier City, La.) and 245 larvae collected on 24 June 1988 (St. Gabriel Research Station, St. Gabriel, La.). These collections consisted of larvae in various instars and were randomly mixed in the laboratory and reared for one generation before testing. The pyrethroid-resistant PY-R tobacco budworm strain was initiated from a colony (PEG-87) maintained at ICI Americas, Inc. (Wilmington, Del.). The PY-R strain represents a combination of larval collections from cotton fields in several states following control failures with pyrethroids. It was continuously selected in each generation with cypermethrin to maintain a stable level of resistance. This strain was received in our laboratory on 18 February 1988 and was not exposed with cypermethrin during the course of this experiment. At initiation of the present study, larvae of the PY-R strain were 200 times more

resistant to cypermethrin than the PY-S strain. The pyrethroid-susceptible PY-S tobacco budworm colony was initiated in 1977 from a collection of larvae obtained from a cotton field in Louisiana (Leonard et al. 1988). The PY-S strain has been maintained in continuous culture at the Department of Entomology, Louisiana State University, Baton Rouge, La. The FIELD-88 strain was established from a collection of ca. 475 larvae taken from a cotton field near Newellton, La., on 9 and 10 June 1988. Larvae of this strain were 6 times more resistant to cypermethrin (F_1 generation) than larvae of the PY-S laboratory strain.

All insect strains were reared in a similar manner according to the procedures described by Leonard et al. (1988). Larvae were reared on a modified pinto bean and wheat germ artificial diet (Shour & Sparks 1981), while adults were fed an aqueous sugar (1:10 sugar:water ratio) solution. These insects were held at a 14:10 (L:D) photoperiod, 28 $\pm$ 3°C and 65-70% RH for the duration of this experiment.

Cotton Lines

The advanced breeding lines selected in this study were chosen based upon field evaluations in Louisiana during 1984-1986 (Jones et al. 1987, Stringer 1987). The two Louisiana breeding lines (La. HG-063 and La. HG-660) have a high frequency of glands on the calyx lobes of the flower

bud that has been associated with increased levels of terpenoids, primarily gossypol (Sappenfield et al. 1974). The two lines originated from a cross made in 1977 between La. HG83-1-1546 and La. HG1838-1497 (Jones et al. 1988). The PD-0804 cotton line was obtained from the cotton breeding program at the Pee Dee Research Center (Pee Dee, S.C.). This line was developed from a cross of two breeding lines, PD 675 and PD 875. The source of resistance in PD-0804 has not been associated with increased levels of terpenoids and is presently unknown. A commercial cultivar, 'Deltapine 41' (DPL 41; Delta Pine and Land Company, Scott, Miss.), was included as a susceptible cultivar.

Plots of land (4 rows on 1.01 m centers by 60.6 m) at the Department of Agronomy (Louisiana State University) research farm, Baton Rouge, La., were planted on 18 May 1988 with seed of each cotton line. Agronomic practices recommended for cotton production on soils with high yield potential in Louisiana were used to maintain plots. Selective insecticides were used to control pests (primarily boll weevil, Anthonomus grandis grandis Boheman) to assure an adequate supply of undamaged flower buds (squares) for the test.

Test Procedures

Cotton squares were harvested three times per week from the field plots. In the laboratory, the squares were

debracted, rinsed with tap water, allowed to air dry and refrigerated. The artificial diet was included as an additional control to compare differences in growth and development between field and laboratory adapted tobacco budworm strains. Weights (Mean ± SE) in g of debracted squares were determined for La. HG-660 (0.53 ± 0.14), La. HG-063 (0.56 ± 0.15), PD-0804 (0.52 ± 0.17), DPL 41 (0.59 ± 0.18) and blocks of artificial diet (1.25 ± 0.16). Larvae (2-3 d old) were removed from each colony and individually placed in plastic cups (29.7 ml) containing two squares from a single cotton line or one block of artificial diet. These cups were sealed with removable cardboard lids. Fresh squares and blocks of artificial diet were introduced daily to their respective cups until the larvae pupated or died. This experiment was conducted within three generations of the FIELD-88 and CORN-BW strains being removed from the field.

The experiment was a randomized complete block design with a factorial arrangement of treatment combinations (insect strains and cotton line/diets). Each treatment combination consisted of 5 cups (one larva/cup) per replicate. The experiment was replicated eight times. Data were collected on larval weights (5 and 9 d after initiation of experiment), pupal weights (1 d after pupation) and cumulative mortality to adult emergence. The

duration of the entire larval and pupal stadia also were recorded. Measurements were recorded for each cup, totaled for each treatment combination within a replicate, and these means were used in the statistical analyses. Results were subjected to an analysis of variance (ANOVA) to determine significant treatment effects. Duncan's (1955) Multiple Range Test was used to separate significant treatment means ($P = 0.05$). These procedures were done using Statistical Analysis Systems (SAS) software for personal computers (SAS Institute 1988).

Results

All insect strains responded similarly to the cotton line/diet treatments (Table 1). No significant interaction between insect strain and cotton line/diet was observed for 5 (F = 1.13; df = 12, 78; P = 0.34) or 9 d larval weights (F = 0.75; df = 12, 75; P = 0.70). However, pupal weights (F = 2.10; df = 12, 74; P = 0.03) were significantly influenced by the interaction between insect strain and the cotton line/diet treatments. Pupal weights of the Corn-BW strain were higher than pupal weights of the tobacco budworm strains when larvae were reared on artificial diet or squares of DPL 41. This interaction occurred as the result of pupal weights of the CORN-BW strain being lower than pupal weights of the FIELD-88 and PY-R strains when larvae were reared on squares of La. HG-660 and La. HG-063. No interaction between these factors was found for larval stadia duration (F = 1.53; df = 12, 47; P = 0.16) and d to adult eclosion (F = 1.85; df = 12, 39; P = 0.09). The mean duration of time required for larvae from the different strains to pupate was extremely variable (13.2 to 21.6 d). Larvae from the different insect strains, that did pupate after an extended period, were associated with low pupal weights and increased pupal mortality. There was no insect strain and cotton line/diet interaction for cumulative

mortality to adult eclosion (F = 1.02; df = 12, 84; P = 0.44).

Differences among insect strains in 5 d larval weights (F = 10.61; df = 3, 78; P < 0.001), 9 d larval weights (F = 22.23; df = 3,75; P < 0.001) and pupal weights (F = 33.01; df = 3, 74; P < 0.001) were highly significant (Table 1). The CORN-BW strain had a significantly higher 5 d larval weight than was observed for the PY-S and PY-R tobacco budworm strains. The CORN-BW strain also had a significantly higher 9 d larval weight than was observed for all tobacco budworm strains. The FIELD-88 tobacco budworm strain had significantly higher larval weights than was detected for the PY-S and PY-R strains and significantly higher pupal weights compared to the PY-S strain. There were no significant differences observed among strains in larval stadia duration (F = 2.04; df = 3, 47; P = 0.12) and d to adult eclosion (F = 0.71; df = 3, 39; P = 0.54). Cumulative mortality among insect strains was found to be significantly different (F = 16.61; df = 3, 84; P < 0.001). The highest mortality occurred in the PY-S and CORN-BW strains and was significantly higher than the mortality observed for the FIELD-88 strain.

Cotton line/diet treatments also had a significant influence on insect development and survival (Fig. 1). Significant differences were observed among treatments for

5 d larval weights (F = 16.51; df = 4, 78; P < 0.001), 9 d larval weights (F = 21.53; df = 4, 75; P < 0.001) and pupal weights (F = 63.21; df = 4, 74; P < 0.001). All cotton line treatments significantly reduced larval (5 and 9 d) and pupal weights compared to larval and pupal weights observed for insects reared on the artificial diet. Larvae reared on the two Louisiana germplasm lines (La. HG-660 and La. HG-063) had lower larval (5 & 9 d) and pupal weights than larvae reared on squares of the other cotton lines. There was no difference in larval (5 and 9 d) and pupal weights between insects reared on the DPL 41 and PD-0804 cotton lines.

Larvae reared on cotton squares required significantly longer time to pupate (F = 40.7; df = 4, 47; P < 0.001) and emerge as adults (F = 13.9; df = 4, 39; P < 0.001) than larvae fed artificial diet (Fig. 2). Larval stadia duration was significantly greater for insects reared on La. HG-660 and La. HG-063 compared to DPL 41 and PD-0804, but d to adult emergence for the two Louisiana cotton lines was not significantly different from that for DPL 41.

Cumulative mortality was also significantly different among cotton line/diet treatments at the 5 d larval (F = 38.82; df = 4, 84; P < 0.001), 9 d larval (F = 57.70; df = 4, 84; P < 0.001), pupal (F = 57.85; df = 4, 84; P < 0.001) and adult (F = 73.99; df = 4, 84; P < 0.001) sample

intervals (Fig. 3). Mortality recorded for insects reared on the artificial diet was significantly lower than mortality observed for larvae reared on the cotton cultivar/lines at all sample intervals. The highest mortality was observed for larvae reared on the La. HG-660 and La. HG-063 cotton lines, which was significantly higher than the mortality recorded for larvae reared on the DPL 41 and PD-0804 cotton lines.

Table 1. Growth, development and survival of pyrethroid-resistant, -susceptible and field strains of tobacco budworm and bollworm on selected cottons and artificial diet

Insect strain	Cotton line/diet	5 d larval wt (mg)	Strain x ± SE	9 d larval wt (mg)	Strain x ± SE	Pupal wt (mg)	Strain x ± SE	D to pupation	Strain x ± SE	D to adult eclosion	Strain x ± SE	Mortality (%)	Strain x ± SE
PY-S	Diet[a]	60.5		207.9		228.4		14.7		24.5		50.0	
	DPL 41	26.0		123.0		120.4		18.1		28.8		77.5	
	PD-0804	37.4	33.8 ± 5.2b	145.5	126.3 ± 14.1c	155.5	122.2 ± 14.6b	16.8	18.1 ± 0.7a	26.3	27.5 ± 0.8a	75.8	72.9 ± 2.4a
	La. HG-063	18.8		72.6		52.0		21.6		30.0		80.6	
	La. HG-660	26.5		82.4		54.8		19.7		28.0		80.6	
FIELD-88	Diet	80.5		275.9		288.7		13.4		24.9		37.5	
	DPL 41	34.0		232.5		188.8		18.8		26.8		52.5	
	PD-0804	53.0	48.3 ± 7.4a	221.7	202.5 ± 15.5b	180.9	169.9 ± 13.3a	18.2	17.2 ± 0.7a	25.3	26.4 ± 0.6a	62.3	59.5 ± 3.3c
	La. HG-063	35.3		138.5		90.1		17.2		27.7		72.5	
	La. HG-660	38.8		143.8		101.0		18.3		27.6		72.7	
PY-R	Diet	54.0		289.3		263.1		14.0		24.4		35.3	
	DPL 41	25.8		132.3		198.2		18.3		28.2		65.0	
	PD-0804	30.5	29.3 ± 3.3b	165.3	148.5 ± 16.5c	176.8	166.6 ± 14.0a	17.6	18.1 ± 0.6a	27.7	27.4 ± 0.7a	65.0	65.3 ± 3.4bc
	La. HG-063	19.0		78.8		98.8		19.9		25.5		80.2	
	La. HG-660	17.4		76.5		96.0		20.6		31.0		80.8	
CORN-BW	Diet	125.4		400.1		362.9		13.2		23.3		45.3	
	DPL 41	51.9		254.4		257.8		17.4		27.5		72.8	
	PD-0804	60.2	60.7 ± 9.7a	303.5	253.8 ± 26.2a	172.5	187.8 ± 23.1a	17.1	17.4 ± 0.7a	24.9	27.3 ± 0.7a	67.8	68.7 ± 2.8ab
	La. HG-063	32.1		146.4		60.1		19.9		30.2		77.4	
	La. HG-660	33.2		164.8		85.8		19.2		30.7		80.4	

Means in a column followed by a common letter are not significantly different ($P < 0.05$; Duncan's [1955] Multiple Range Test).

[a] Modified pinto bean and wheat germ artificial diet (Shour & Sparks 1981).

FIG. 1. Larval (5 & 9 d) and pupal (1 d) weights (mean ± SE) of tobacco budworm and bollworm strains reared on selected cotton lines and artificial diet.

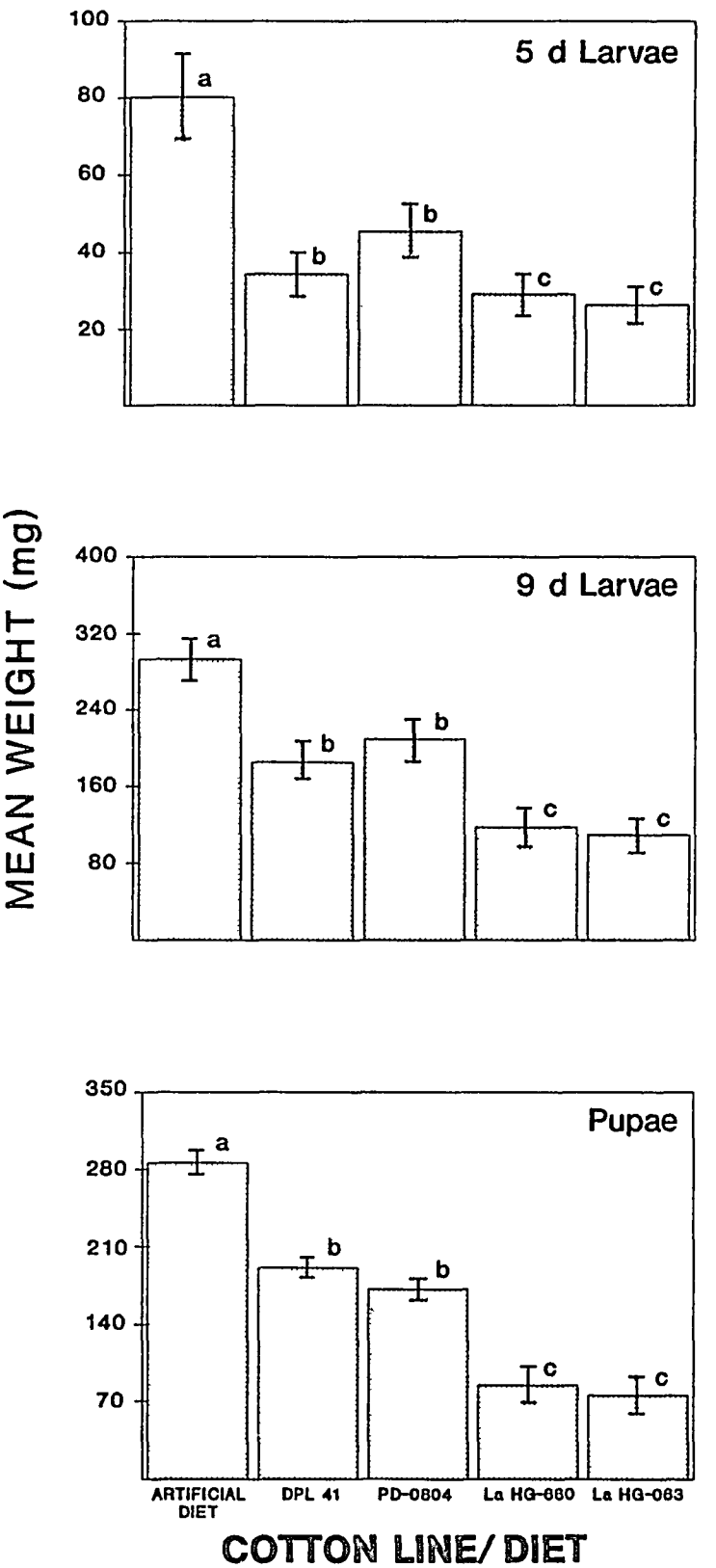

5 d Larvae
9 d Larvae
Pupae
MEAN WEIGHT (mg)
100
80
60
40
20
400
320
240
160
80
350
280
210
140
70
a
b
b
c
c
ARTIFICIAL DIET
DPL 41
PD-0804
La HG-660
La HG-663
COTTON LINE/ DIET

FIG. 2. Effect of selected cotton lines and artificial diet on number of days (mean ± SE) required for tobacco budworm and bollworm strains to pupate and eclose as adults.

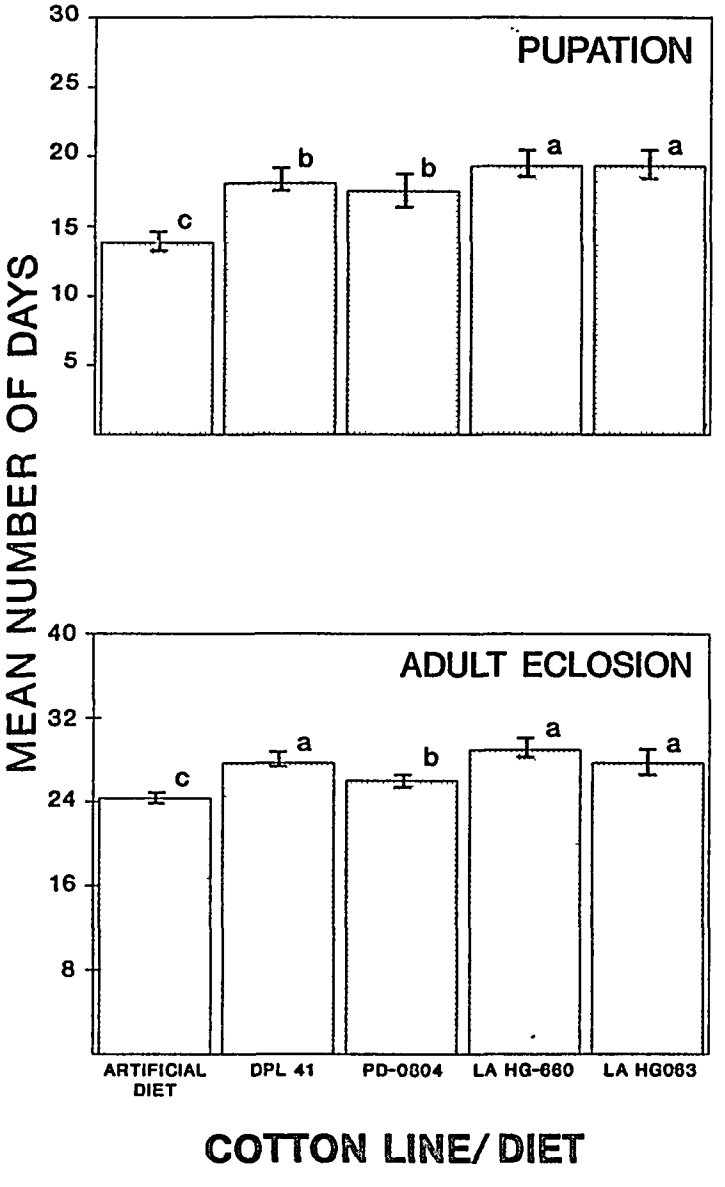
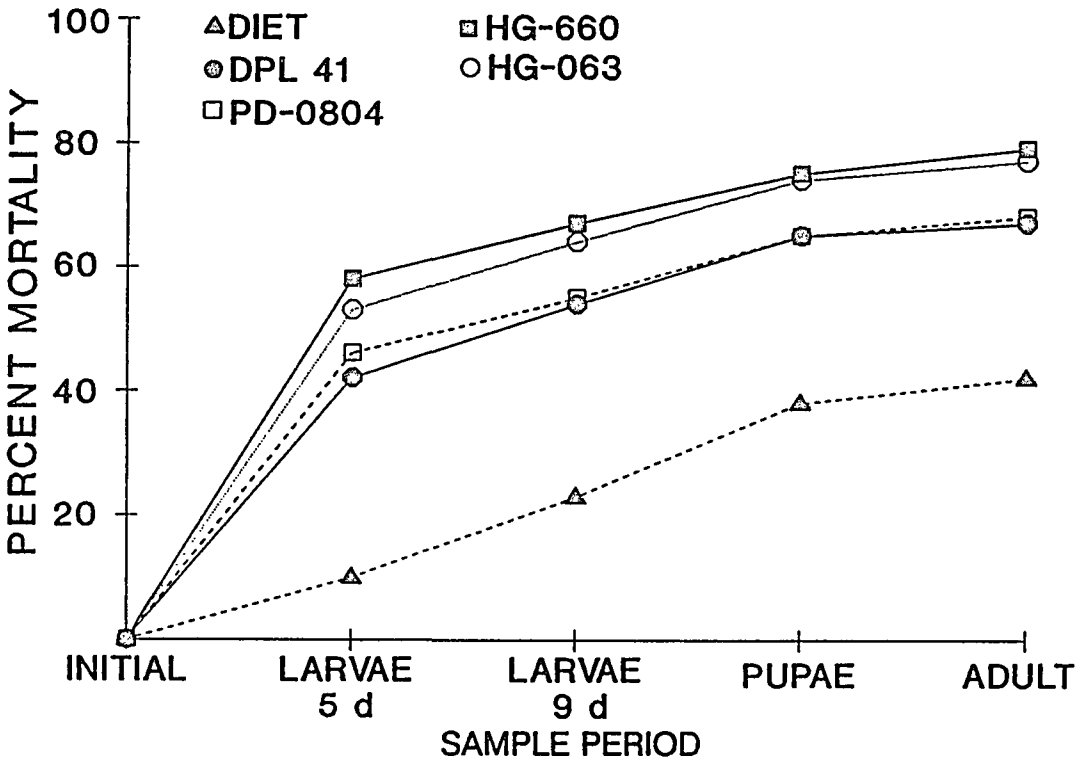

PUPATION
ADULT ECLOSION
MEAN NUMBER OF DAYS
30
25
20
15
10
5
40
32
24
16
8
a
b
c
ARTIFICIAL DIET
DPL 41
PD-0604
LA HG-660
LA HG063
COTTON LINE/ DIET

FIG. 3. Cumulative mortality of tobacco budworm and bollworm strains at different developmental stages influenced by larval rearing on different cotton lines and artificial diet.


100
80
60
40
20
PERCENT MORTALITY
△DIET
▫HG-660
◉DPL 41
○HG-063
□PD-0804
INITIAL
LARVAE 5 d
LARVAE 9 d
PUPAE
ADULT
SAMPLE PERIOD

Discussion

Differences in the development and survival of the different insect strains appeared to be more related to the adaptation to laboratory rearing and the duration of time removed from the field rather than to the level of pyrethroid susceptibility. Although the PY-R strain has been shown to have increased levels of mixed function oxidase and esterase enzyme activity relative to pyrethroid -susceptible strains (Dowd et al. 1987, Little et al. 1989, McCaffery et al. 1989), this apparently did not influence the sensitivity of the larvae to the different cotton lines. The PY-R strain does not have an enhanced ability to survive on the Louisiana cotton lines although evidence suggests that several cotton allelochemicals (gossypol, tannins and isoquercitrin) are detoxified via the mixed function oxidase enzyme system in tobacco budworm (Mullin 1985, Hedin et al. 1988). In another study, gossypol was the only one of five allelochemicals (gossypol, umbelliferone, scopeletin, ß-caryophyllene and [+]-α-pinene) which had little or no effect on induction of cytochrome P-450 content and associated metabolic activity in tobacco budworm (Brattsten 1987b).

Larvae of the FIELD-88 and CORN-BW field strains gained more weight when reared on the cotton lines than the other

tobacco budworm strains. The PY-S and PY-R strains have been cultured in the laboratory for multiple generations (> 100 and > 20 generations, respectively) with larvae being reared on artificial diet. These two laboratory strains, especially the PY-S laboratory strain, may not be able to metabolize allelochemicals in the cotton lines. Furthermore, the highest cumulative mortality was observed for the laboratory adapted PY-S strain.

The two Louisiana germplasm cotton lines, La. HG-660 and La. HG-063, significantly suppressed larval and pupal weight gain while increasing the larval stadia duration compared to larvae reared on other cotton lines. The significant interaction between the CORN-BW strain and La. breeding lines for pupal weight indicate that bollworms may be more sensitive to the resistance traits in these cotton lines than are the tobacco budworm strains. These two cotton lines also caused higher cumulative mortality throughout the larval, pupal and adult stages of development for all strains. In a related study, Montandon et. al. (1987) concluded that the reduction in growth of tobacco budworm larvae was due to a reduced efficiency of food conversion into biomass and not due to reduced consumption. The reduction in growth and survival, as well as the delay in development observed for larvae fed squares of La. HG-660 and La. HG-063 may result from antixenosis as well as

antibiosis. Parrott et al. (1983) found small tobacco budworm larvae avoid feeding on or around the glands on the square. Increased glandulosity on the flower bud reduces the preferred feeding sites and would relate directly to reduced consumption.

Larvae fed squares of PD-0804 had similar weights, developmental times and survival as compared to those larvae fed squares of the commercial cultivar, DPL 41. These data are in direct contrast to results observed in field trials that demonstrated reduced levels of feeding damage and increased yields compared to damage and yields of commercial cultivars susceptible to tobacco budworm and bollworm (Jones et al. 1985, 1986). However, the source of resistance in PD-0804 may be a water soluble compound, and it could have been removed during the preparation of the squares for larval feeding. Bhardwaj et al. (1985) demonstrated a significant increase in bollworm larval growth by washing plant terminals of PD-675 compared to growth of larvae fed unwashed plant terminals. The PD-675 resistant line was one of the original parents of PD-0804 and the mechanism of resistance may be similar. Furthermore, PD-675 has been reported to be more susceptible to bollworm during years with extended periods of rainfall than in years with less rainfall (Bhardwaj et al. 1985).

The incidence of mortality between the initiation of

the test and the 5 d larval weight sample was much higher than the level of mortality observed during other sample intervals. Although most of the larval mortality in the early instars is the result of diet effects through either antibiosis or antixenosis, a portion of the observed mortality may be associated with the handling of larvae during daily feeding. However, other studies support the higher susceptibility of earlier instar larvae to cotton allelochemicals relative to later instars (Shaver & Parrot 1970, Montandon et al. 1987).

Plant resistance in cotton and insecticide use have generally been considered as compatible tools for cotton insect management. However, development of insecticide resistance in tobacco budworm could negatively impact cotton plant resistance by increasing the capacity of insects' to metabolize plant allelochemicals. In this study, prior selection with pyrethroids against a laboratory strain of tobacco budworm did not enhance development and survival of larvae fed resistant cotton lines, although similar enzyme systems have been implicated in the detoxification of pyrethroids and terpenes such as gossypol. This lack of association may have been, in part, due to the qualitative nature of the enzyme systems involved, the compounds being detoxified and the length of time a pyrethroid-resistant strain had been removed from the field. Considering the

variety and quantity of insecticides used on cotton, the possibility of preadaptation of insect species, especially tobacco budworm, to allelochemicals in cotton should be considered in future studies.

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CHAPTER II

MANAGEMENT OF TOBACCO BUDWORM AND BOLLWORM IN COTTON UTILIZING PLANT RESISTANCE IN COMBINATION WITH SELECTED INSECTICIDES

Introduction

Integrating cotton varietal resistance with chemical control to manage tobacco budworm, Heliothis virescens (F.), and bollworm, Helicoverpa zea (Boddie), has been suggested by previous researchers (Lincoln et al. 1971, Parrott et al. 1973, Culp et al. 1978), but relatively few studies have been conducted to examine the potential of such an interaction. Several advances in cotton plant resistance to tobacco budworm and bollworm have been reported (Bell & Stipanovic 1977, Hedin et al. 1983, Schneider et al. 1986, Stringer 1987, Benedict et al. 1988), but until recently plant resistance has not been emphasized because of the cost effectiveness of the presently used insecticides. In general, the efficacy of varietal resistance has been unable to compete with the degree of control observed with insecticides against these cotton pests. However, as pyrethroid resistance in tobacco budworm continues to develop and become more widespread, additional management tactics will be necessary to control this pest.

Studies that have evaluated the interaction of cotton varietal traits and insecticide toxicity to tobacco budworm and bollworm have generally yielded promising results. Field studies have demonstrated that the use of partially resistant cultivars in combination with insecticides has

resulted in control of these pests equal to or better than the commercially recommended strategies (Lincoln et al. 1971, Schuster & Anderson 1976, Culp et al. 1978). In most cases, these experiments evaluated insecticidal efficacy as affected by different morphological characteristics of cotton varieties. Laboratory studies have demonstrated either no effect or decreases in insecticide toxicity to noctuid pests, including tobacco budworm and bollworm, when allelochemicals derived from cotton plants were added to artificial rearing media (Perkins & Canerday 1971, Abou-Donia et al. 1974, Shaver & Wolfenbarger 1976). Other studies have shown that tobacco budworm and bollworm larvae reared on flower buds of cotton lines containing allelochemicals are more sensitive to some insecticides (Mullins 1980, Muehleisen et al. 1989). The interactions of the cotton plant allelochemicals and insecticide susceptibility of these pests remains unclear. Of the plant compounds identified in cotton, gossypol and related terpenoids have been more extensively studied as a source of chemical resistance against tobacco budworm and bollworm (Bell & Stipanovic 1977, Hedin et al. 1983, Schneider et al. 1986, Stringer 1987).

Because the tobacco budworm is developing resistance to the pyrethroids and the potential of alternative insecticides appears limited, additional approaches to

managing this pest should be investigated. At the present time, cotton plant resistance cannot be relied upon as a single tool in a management system, but perhaps it can be successfully used in combination with selected chemical controls. Thus, this study was initiated to determine the interaction of one potential source of cotton varietal resistance (high gossypol) with selected insecticide treatments on tobacco budworm and bollworm infestations. The effects of selected cotton cultivar/lines and insecticide combinations on insect infestations, cotton yield and associated agronomic properties were determined.

Methods and Materials

Field trials were conducted at the Dean Lee Research Station, Alexandria, La., during 1988 and at the Northeast Research Station, St. Joseph, La., during 1989. The two Louisiana germplasm lines (La. HG-660 and La. FNHG-850075) were provided by the Department of Agronomy, Louisiana Agricultural Experiment Station, Louisiana State University Agricultural Center, Baton Rouge, La. The lines were selected based on favorable agronomic qualities and field trial results during 1984-1986 showing resistance to tobacco budworm and bollworm (Jones et al. 1987, 1988, 1989). The flower buds of these cotton lines contain a high frequency of glands on the calyx lobes of the flower bud. Sappenfield et al. (1974) had previously associated these glands with increased levels of terpenoids, primarily gossypol. The La. HG-660 line was derived from a cross between two high gossypol strains, La. HG 83-1-1546 and La. HG 1838-1497 (Jones et al. 1988). The two parent strains were selected from an intercrossed population involving a commercial cultivar, 'Stoneville 213', and a Louisiana advanced cotton strain, GT5A-10-15-2XG15 (Jones et al. 1988).

Standard agronomic and weed control practices for cotton production in these two regions of Louisiana were used to maintain the plots. In furrow applications of

aldicarb 15G (0.56 kg [AI]/ha) and terraclor + terrazole (1.1 + 0.28 kg [AI]/ha) were applied at planting to manage early season insects and seedling diseases.

Infestation and damage ratings for tobacco budworm, bollworm and boll weevil, Anthonomus grandis grandis Boheman were determined by examining 50 flower buds (cotton squares) and 25 plant terminals from the center two rows of each plot on several sample dates. Plant maturity and boll density were estimated by determining the total number of bolls on a 3.3 m section of row randomly chosen from the center two rows of each plot. The number of harvestable bolls were partitioned into those that had opened and those that remained closed to estimate differences in maturity among treatments. Yields were estimated by mechanically harvesting the four inside rows of each plot in 1988 and the two inside rows of each plot in 1989 with a John Deere two row spindle-type cotton picker. Handpicked samples, consisting of 25 randomly chosen bolls from the center two rows of each plot, were transported to the laboratory to determine boll weight, lint:seed ratio and fiber quality.

1988 Dean Lee Research Station Trial

Cotton seed of a susceptible cultivar, 'Deltapine 41' (DPL 41) and La. HG-660, were planted on 12 May in a Norwood silt loam. Treatments were assigned to field plots in a factorial arrangement within a randomized complete block

design and replicated four times. Plots consisted of eight rows on 0.96 m centers and 28.8 m long. The insecticide treatments evaluated in this study included lambda-cyhalothrin 1E (0.028 kg [AI]/ha) and thiodicarb 3.2F (0.28 kg [AI]/ha), in addition to an untreated control for each cultivar/line. Treatments were applied using a John Deere high clearance sprayer calibrated to deliver 57 liters total spray solution per ha. Insecticides were applied on 14, 18, 25 July, on 1, 16, 22 August and on 1 September. All plots were oversprayed with azinphosmethyl 2E (0.28 kg [AI]/ha), methyl parathion 4E (0.28 kg [AI]/ha) or malathion 4.1E (0.9 kg [AI]/ha) on 14, 18, 25 July, on 1, 12, 16, 22 August and on 1, 7 September to suppress boll weevil populations. Methamidophos 4E (0.34 kg [AI]/ha) was oversprayed on the test area as an aphicide on 16 August. Treatments were evaluated on 18, 22, 28 July and on 1, 5, 18 August. Additional tobacco budworm and bollworm damage ratings were made by sampling 50 bolls per plot on 26 August and on 7 September. Data were also collected for treatment effects on soybean looper, Pseudoplusia includens (Walker), infestations in the plots on 7 September by recording an infestation rating (IR) as 1 (no feeding), 2 (feeding in the lower portion of the plant) or 3 (feeding throughout the plant). Plots were defoliated on 4 October using DEF 6 + methyl parathion 4E (1.35 + 0.28 kg [AI]/ha). Plant

maturity and boll density were estimated on 21 September. Handpicked samples were collected and plots were mechanically harvested on 13 October.

1989 Northeast Research Station Trial

Cotton cultivar/line seed of DPL 41, La. HG-660 and La. FNHG-850075, were planted on 15 May and replanted on 24 May to ensure an adequate stand. Treatments were assigned to field plots in a randomized complete block design and replicated four times. Plots consisted of four rows on 0.96 m centers and 19.5 m in length. The insecticide treatments evaluated in this study included lambda-cyhalothrin 1E (0.028 kg [AI]/ha), thiodicarb 3.2F (0.28 kg [AI]/ha), Bacillus thuringiensis var. Kurstaki (Dipel ES, 1.8 l/ha formulated material) and thiodicarb (0.28 kg [AI]/ha) + Dipel ES (1.8 liters/ha formulated material), in addition to an untreated control for each cultivar/line. Treatments were applied using a John Deere high clearance sprayer calibrated to deliver 47 liters total spray solution per ha. Insecticides were applied on 19, 24, July and on 2, 25, 28 August. All plots were oversprayed with azinphosmethyl 2E (0.28 kg [AI]/ha), methyl parathion 4E (0.28 kg [AI]/ha) or malathion 4.1E (0.9 kg [AI]/ha) on 19, 25 July, on 4, 8, 14, 17, 19, 23, 28 August and on 1 September to suppress boll weevil populations. Methamidophos 4E (0.34 kg [AI]/ha) or endosulfan 2E (0.56 kg [AI]/ha) was applied on 12, 19 July

and on 17, 23, 28 August to suppress cotton aphids, Aphis gossypii Glover. Treatments were evaluated on 27 July and on 4, 10 August. Additional tobacco budworm and bollworm damage ratings were made by sampling 50 bolls per plot on 28 August. Plant maturity and boll density were estimated on 21 September. Plots were defoliated on 2 October using DEF 6 + methyl parathion 4E (1.35 + 0.28 kg [AI]/ha). Plots were mechanically harvested and hand picked samples were collected on 12 October.

Data Analyses

All data were subjected to analysis of variance using the general linear model (GLM) procedure of statistical analysis systems adapted for the microcomputer (SAS Institute 1988). Treatment means were separated using Duncan's (1955) multiple range test ($P = 0.05$).

Results

1988 Dean Lee Research Station Trial

There was no significant interaction ($P < 0.05$) between cotton cultivar/line and insecticide treatments on all variables in this experiment. Significant effects on variables were attributed to either cotton cultivar/line or insecticide treatment.

Numbers of tobacco budworm and bollworm eggs in the plots were not significantly influenced by cultivar/line ($F = 2.98$; df = 1,70; $P = 0.105$) in 1988 (Table 1). Plots planted to La. HG-660 had significantly fewer larvae in plant terminals ($F = 36.34$; df = 1,70; $P < 0.001$), damaged plant terminals ($F = 6.05$; df = 1,70; $P = 0.027$), larval damaged squares ($F = 39.27$; df = 1,70; $P < 0.001$) and larval damaged bolls ($F = 4.08$; df = 1,23; $P = 0.050$) than were observed in the plots planted to DPL 41. Cotton cultivar/line had no significant effect on numbers of boll weevil damaged squares ($F = 0.82$; df = 1,70; $P = 0.381$) in this trial. Also, cultivar/line had no significant effect on soybean looper defoliation ratings ($F = 0.25$; df = 1,15; $P < 0.626$). Plots planted to La. HG-660 had an IR of 1.2 compared to an IR of 1.4 for plots planted to DPL 41.

Numbers of tobacco budworm and bollworm eggs in the plots also were significantly influenced by insecticide

treatments (F = 2.10; df = 2,70; P = 0.157) in this experiment (Table 2). The insecticide treatments significantly influenced numbers of damaged plant terminals (F = 14.23; df = 2,70; P < 0.001), damaged squares (F = 19.08; df = 2,70; P < 0.001) and damaged bolls (F = 11.27; df = 2,23; P < 0.001). Both lambda-cyhalothrin and thiodicarb significantly reduced the number of damaged plant terminals, damaged squares and damaged bolls compared to numbers in the untreated plots. The lambda-cyhalothrin treated plots had significantly fewer damaged squares than was observed in the thiodicarb treated plots. Insecticide treatments had no significant effect on numbers of larvae in plant terminals (F = 3.02; df = 2,70; P = 0.079) or boll weevil infestations (F = 0.15; df = 2,70; P = 0.865) in the plots. There were significant differences in soybean looper defoliation (F = 4.46; df = 2,15; P = 0.030) among insecticide treated plots. Damage levels observed in the lambda-cyhalothrin treated plot (IR = 0.6) were significantly lower than the damage levels in the thiodicarb treated (IR = 1.6) and untreated (IR = 1.7) plots.

Plots planted to La. HG-660 had significantly more total bolls (F = 4.42; df = 1,15; P = 0.051), and matured significantly earlier (F = 9.74; df = 1,15; P = 0.007) than the plots planted to DPL 41 (Table 3). However, there was no significant difference in seed cotton yields (F = 1.05;

df = 1,15; P = 0.321) and lint (F = 4.08; df = 1,15; P = 0.061) between the La. HG-660 and DPL 41 plots. Boll weights and lint turnout were significantly higher in the La. HG-660 plots (F = 51.96; df = 1,15; P < 0.001 and F = 108.99; df = 1,15; P < 0.001, respectively) compared to the DPL 41 plots.

Insecticide treatments had no significant effect on total numbers of bolls (F = 2.16; df = 2,15; P = 0.150), crop maturity (F = 0.60; df = 2,15; P = 0.563), lint turnout (F = 2.18; df = 2,15; P = 0.149) or boll weight (F = 0.53; df = 2,15; P = 0.601) (Table 4). However, the insecticide treated plots yielded significantly more seed cotton (F = 20.25; df = 2,15; P < 0.001) and lint (F = 18.90; df = 2,15; P < 0.001) than was produced in the untreated plots. Furthermore, the lambda-cyhalothrin treated plots yielded significantly more seed cotton and lint than did the thiodicarb treated plots.

Cotton cultivar/line had no significant effect on fiber length (F = 0.64; df = 1,15; P = 0.437) or micronaire (F = 1.39; df = 1,15; P = 0.258), but fiber strength was significantly lower (F = 5.03; df = 1,15; P = 0.049) for lint samples of La. HG-660 plots compared to lint samples of DPL 41 (Table 5).

Insecticide treatments had no significant effect on micronaire (F = 1.56; df = 2,15; P = 0.243), fiber strength

(F = 0.19; df = 2,15; P = 0.832) or fiber length (F = 3.39; df = 2,15; P = 0.061) of handpicked cotton samples (Table 6).

1989 Northeast Research Station Trial

Tobacco budworm and bollworm pressure in the test plots was low during most of the 1989 season. There were no significant treatment effects on numbers of larvae in plant terminals (F = 0.68; df = 7,42; P = 0.690), larval damaged squares (F = 1.97; df = 7,42; P = 0.108) and percent larval damaged bolls (F = 1.29; df = 7,42; P = 0.301) in this field trial (Table 7). Significant differences among treatments were observed for numbers of larval damaged terminals (F = 3.48; df = 7,42; P = 0.012) and boll weevil damaged squares (F = 3.17; df = 7,42; P = 0.019). The La. FNHG-850075 plots treated with Dipel ES had significantly fewer damaged plant terminals than was observed in the DPL 41 (treated and untreated) plots. Plots of La. HG-660 (treated and untreated) and La. FNHG-850075 (treated and untreated) had significantly fewer damaged plant terminals compared to the number in the DPL 41 untreated plots. Plots planted to La. FNHG-850075 (treated and untreated) had significantly fewer boll weevil damaged squares compared to the number in the untreated DPL 41 and untreated La. HG-660 plots.

No significant differences were observed among treatments in numbers of total bolls (F = 0.53; df = 7,21;

P = 0.806) sampled from the plots (Table 8). However, there were significant differences among treatments in crop maturity (F = 2.58; df = 7,21; P = 0.044), seed cotton yields (F = 2.81; df = 7,21; P = 0.031), lint yields (F = 2.63; df = 7,21; P = 0.040), lint turnout (F = 23.91; df = 7,21; P < 0.001) and boll weights (F = 3.85; df = 7,21; P = 0.008). The La. HG-660 (untreated) plots matured significantly earlier than the La. FNHG-850075 (treated and untreated) plots based on percent open bolls. The La. FNHG-850075 plot treated with thiodicarb yielded significantly more seed cotton than the untreated DPL 41 plot, the La. HG-660 plot treated with thiodicarb and the other La. FNHG-850075 plots. The lint yield of the La. FNHG-850075 plot treated with thiodicarb yielded significantly more seed cotton than the untreated DPL 41 plot, the HG-660 plot treated with thiodicarb, the La. FNHG-850075 plot treated with Dipel ES and the La. FNHG-850075 plot treated with Dipel ES + thiodicarb. Samples of cotton from the DPL 41 plots (untreated and treated) had a significantly higher lint turnout compared to the lint turnout of all other treatments (Table 8). Boll weights of DPL 41 (treated and untreated) were significantly higher than boll weights of La. HG-660 (treated and untreated).

The cultivar/line and insecticide treatment combinations had no significant effect on fiber length (F

= 2.28; df = 7,21; P = 0.068), micronaire (F = 1.71; df = 7,21; P = 0.161) and fiber strength (F = 1.49; df = 7,21; P = 0.223) of lint samples (Table 9).

Table 1. Effect of cotton cultivar/line and insecticide treatment combinations on tobacco budworm and bollworm larval infestations and boll weevil damage during 1988

Management Components		No. per 50 terminals						No. per 50 fruiting structures					
Cultivar	Insecticide	Eggs[1]	Cultivar $\bar{x}$ ± SE	Larvae	Cultivar $\bar{x}$ ± SE	Larval[a] Damaged Term.	Cultivar $\bar{x}$ ± SE	Larval[a] Damaged Squares	Cultivar $\bar{x}$ ± SE	Weevil[a] Damaged Squares	Cultivar $\bar{x}$ ± SE	Larval[b] Damaged Bolls	Cultivar $\bar{x}$ ± SE
DPL 41	Untreated	2.7		2.9		14.4		3.7		1.8		5.3	
	L-cyhalothrin	4.1	3.4 ± 0.4a	1.3	2.1 ± 0.3a	8.1	10.8 ± 0.4a	0.8	2.2 ± 0.2a	1.3	1.6 ± 0.2a	1.8	3.2 ± 0.5a
	Thiodicarb	3.4		2.0		9.9		2.1		1.7		2.5	
La. HG-660	Untreated	2.8		1.4		9.0		1.5		1.8		3.3	
	L-cyhalothrin	3.4	3.0 ± 0.4a	0.6	1.1 ± 0.3b	5.2	7.1 ± 0.6b	0.5	1.2 ± 0.3b	1.6	1.7 ± 0.2a	1.1	2.2 ± 0.3b
	Thiodicarb	2.7		1.2		7.2		1.5		1.8		2.1	

Means within columns for cultivar/lines followed by a common letter are not significantly different ($P > 0.05$, Duncan's [1955] multiple range test).

[a] Values represent seasonal means derived from analysis across five sample dates.

[b] Values represent seasonal means derived from analysis across two sample dates.

Table 2. Effect of insecticide treatments on tobacco budworm and bollworm larval infestations and boll weevil damage during 1988 (± SE)

	No. per 50 Terminals			No. per 50 Fruiting Forms		
Treatments	Eggs[a]	Larvae[a]	Larval[a] Damaged Terminals	Larval[a] Damaged Squares	Weevil[a] Damaged Squares	Boll[b] Damage
Untreated	2.7 ± 0.5a	2.2 ± 0.3a	11.7 ± 1.0a	2.8 ± 0.5a	1.8 ± 0.3a	4.3 ± 0.5a
L-cyhalothrin	3.7 ± 0.6a	0.9 ± 0.4a	6.6 ± 0.6b	0.7 ± 0.3c	1.5 ± 0.2a	1.4 ± 0.5b
Thiodicarb	3.0 ± 0.4a	1.6 ± 0.4a	8.5 ± 1.0b	1.8 ± 0.3b	1.8 ± 0.2a	2.3 ± 0.3b

Means within columns followed by a common letter are not significantly different ($P > 0.05$, Duncan's [1955] multiple range test).

[a] Values represent seasonal means derived from analysis across five sample dates.

[b] Values represent seasonal means derived from analysis across two sample dates.

Table 3. Effect of cotton cultivar/line and insecticide combinations on earliness and cotton yields during 1988

Management Components						Harvested Yield							
Cultivar	Insecticide	Total Bolls[a]	Cultivar $\bar{x}$ ± SE	Percent Open Bolls	Cultivar $\bar{x}$ ± SE	kg SC/ha[b]	Cultivar $\bar{x}$ ± SE	kg Lint/ha	Cultivar $\bar{x}$ ± SE	Lint Turnout	Cultivar $\bar{x}$ ± SE	Boll Wt.(g)	Cultivar $\bar{x}$ ± SE
DPL 41	Untreated	178.0		42.8		2439		1046.6		42.8		5.6	
	L-cyhalothrin	211.0	192.9 ± 8.9b	49.0	44.4 ± 2.0b	2964	2677 ± 67.1a	1286.9	1160 ± 40.0a	43.5	43.3 ± 0.4a	5.4	5.4 ± 0.2a
	Thiodicarb	189.8		41.3		2626		1148.4		43.6		5.4	
La. HG-660	Untreated	207.8		59.1		2561		1029.8		40.1		4.8	
	L-cyhalothrin	298.5	245.7 ± 18.9a	64.3	59.4 ± 2.9a	2944	2737 ± 55.3a	1189.1	1106 ± 24.6a	40.5	40.3 ± 0.2b	4.8	4.7 ± 0.2b
	Thiodicarb	230.8		54.9		2708		1099.5		40.5		4.4	

Means within columns for each cultivar followed by a common letter are not significantly different ($P > 0.05$, Duncan's [1955] multiple range test).

[a] Number of bolls determined by sampling 3.3 m on one of the two center rows of each plot.

[b] SC refers to seed cotton.

Table 4. Effect of insecticide treatments on earliness and cotton yields during 1988 (± SE)

Treatments	Total Bolls[a]	Percent Open Bolls	Harvested Yield			
			kg SC/ha[b]	kg Lint/ha	Lint Turnout	Boll Wt.(g)
Untreated	192.9 ± 13.0a	51.0 ± 4.0a	2501 ± 55.9c	1038 ± 25.4c	41.4 ± 0.5a	5.2 ± 0.2a
L-cyhalothrin	254.8 ± 36.8a	56.7 ± 5.1a	2954 ± 38.1a	1218 ± 24.5a	42.0 ± 0.6a	5.1 ± 0.2a
Thiodicarb	210.3 ± 11.8a	48.7 ± 3.2a	2667 ± 41.1b	1124 ± 18.0b	42.1 ± 0.5a	4.9 ± 0.3a

Means within columns followed by a common letter are not significantly different ($P > 0.05$, Duncan's [1955] multiple range test).

[a] Number of bolls determined by sampling 3.3 m on one of the two center rows of each plot.

[b] SC refers to seed cotton.

Table 5. Effect of cotton cultivar/line and insecticide combinations on cotton fiber properties during 1988

Management Components		Length	Cultivar	Micronaire	Cultivar	Strength	Cultivar
Cultivar	Insecticide	2.5% SL (cm)	$\bar{x}$ ± SE	Value	$\bar{x}$ ± SE	T_1 g/Tex	$\bar{x}$ ± SE
DPL 41	Untreated	2.934		4.79		24.4	
	L-cyhalothrin	2.946	2.935 ± 0.009a	4.80	4.77 ± 0.04a	24.2	24.2 ± 0.21a
	Thiodicarb	2.926		4.72		24.1	
La. HG-660	Untreated	2.916		4.69		23.4	
	L-cyhalothrin	2.943	2.922 ± 0.025a	4.80	4.70 ± 0.05a	23.3	23.5 ± 0.22b
	Thiodicarb	2.903		4.60		23.8	

Means within columns for each cultivar followed by a common letter are not significantly different ($P > 0.05$, Duncans [1955] multiple range test).

Table 6. Effect of insecticide treatments on cotton fiber properties during 1988 (± SE)

Treatments	Length 2.5% SL (cm)	Micronaire Value	Strength T_1 g/Tex
Untreated	2.925 ± 0.011a	4.74 ± 0.05a	23.9 ± 0.26a
L-cyhalothrin	2.944 ± 0.015a	4.80 ± 0.07a	23.7 ± 0.31a
Thiodicarb	2.915 ± 0.023a	4.66 ± 0.06a	23.9 ± 0.32a

Means within columns followed by a common letter are not significantly different ($P > 0.05$, Duncan's [1955] multiple range test).

Table 7. Effect of cotton cultivar/line and insecticide combinations on tobacco budworm and bollworm larval infestations and boll weevil damage during 1989 (± SE)

Cotton Cultivar/line + Insecticide	No. Per 50 Terminal or Fruit Samples [a]				% Boll Damage
	Terminal Larvae	Larval Damaged Terminals	Larval Damaged Squares	Weevil Damaged Squares	
DPL 41	0.6 ± 0.3a	6.6 ± 0.6a	0.8 ± 0.2a	6.0 ± 1.1ab	1.0 ± 0.5a
DPL 41 + L-cyhalothrin	0.8 ± 0.4a	4.5 ± 1.0ab	0.6 ± 0.2a	3.5 ± 0.7abcd	0.0 ± 0.0a
La. HG-660	0.1 ± 0.0a	3.6 ± 0.6bc	1.0 ± 0.4a	6.3 ± 0.9a	0.5 ± 0.2a
La. HG-660 + Thiodicarb	0.4 ± 0.2a	3.6 ± 0.8bc	0.6 ± 0.4a	5.1 ± 0.8abc	0.0 ± 0.0a
La. FNHG-850075	0.6 ± 0.2a	3.6 ± 0.6bc	0.4 ± 0.2a	1.5 ± 0.4d	0.2 ± 0.1a
La. FNHG-850075 + Thiodicarb	0.1 ± 0.1a	3.0 ± 0.6bc	0.0 ± 0.0a	2.3 ± 0.6cd	0.3 ± 0.1a
La. FNHG-850075 + Dipel ES	0.1 ± 0.0a	1.6 ± 0.8c	0.0 ± 0.0a	1.6 ± 0.3d	0.4 ± 0.2a
La. FNHG-850075 + Thiodicarb + Dipel ES	0.4 ± 0.2a	3.2 ± 0.6bc	0.6 ± 0.3a	1.8 ± 0.5cd	0.3 ± 0.2a

Means within columns followed by a common letter are not significantly different ($P > 0.05$, Duncan's [1955] multiple range test).

[a] Values represent seasonal means derived from analysis across three dates.

Table 8. Effect of cotton cultivar/line and insecticide combinations on earliness and cotton yields during 1989 (± SE)

Cotton Cultivar/line + Insecticide	Total Bolls[a]	Percent Open Bolls	Harvested Yield			
			kg SC/ha[b]	kg Lint/ha	Percent Lint Turnout	Boll Wt.(g)
DPL 41	168.0 ± 49.9a	70.4 ± 10.1ab	2288 ± 620.5b	892 ± 286.7b	39.2 ± 1.0a	4.9 ± 0.2a
DPL 41 + L-cyhalothrin	150.5 ± 21.7a	67.8 ± 9.4ab	2617 ± 755.7ab	1073 ± 341.6a	41.4 ± 0.3a	5.0 ± 0.2a
La. HG-660	209.8 ± 35.5a	76.8 ± 8.1a	2739 ± 708.1ab	959 ± 288.1ab	35.7 ± 0.4b	4.0 ± 0.2c
La. HG-660 + Thiodicarb	171.8 ± 31.0a	71.6 ± 12.7ab	2300 ± 523.7b	828 ± 240.7b	36.1 ± 0.8b	4.2 ± 0.3bc
La. FNHG-850075	164.3 ± 43.5a	54.5 ± 8.9b	2531 ± 692.0b	911 ± 291.1ab	36.6 ± 0.4b	4.8 ± 0.1ab
La. FNHG-850075 + Thiodicarb	180.3 ± 31.2a	56.8 ± 8.2b	3124 ± 899.1a	1094 ± 377.6a	35.1 ± 0.2b	4.4 ± 0.2bc
La. FNHG-850075 + Dipel ES	155.0 ± 40.7a	51.1 ± 13.8b	2559 ± 844.0b	896 ± 361.1b	35.0 ± 0.6b	4.7 ± 0.3ab
La. FNHG-850075 + Thiodicarb + Dipel ES	168.0 ± 47.6a	51.8 ± 16.8b	2356 ± 586.1b	825 ± 241.4b	35.4 ± 0.9b	4.7 ± 0.2ab

Means within columns followed by a common letter are not significantly different ($P > 0.05$, Duncan's [1955] multiple range test).

[a] Number of bolls determined by sampling 3.3 m on one of the two center rows of each plot.

[b] SC refers to seed cotton.

Table 9. Effect of cotton cultivar/lines and insecticide combinations on fiber properties during 1989 (± SE)

Cotton Cultivar/line + Insecticide	Length 2.5% SL (cm)	Micronaire Value	Strength T_1 g/Tex
DPL 41	2.794 ± 0.086a	4.01 ± 0.12a	26.7 ± 0.56a
DPL 41 + L-cyhalothrin	2.839 ± 0.071a	3.81 ± 0.12a	27.2 ± 0.49a
La. HG-660	2.856 ± 0.056a	3.41 ± 0.12a	26.9 ± 0.77a
La. HG-660 + Thiodicarb	2.845 ± 0.061a	3.73 ± 0.23a	27.7 ± 1.08a
La. FNHG-850075	2.908 ± 0.084a	4.05 ± 0.08a	28.1 ± 1.22a
La. FNHG-850075 + Thiodicarb	2.896 ± 0.069a	3.66 ± 0.27a	29.5 ± 1.48a
La. FNHG-850075 + Dipel ES	2.954 ± 0.056a	3.96 ± 0.17a	29.0 ± 1.35a
La. FNHG-850075 + Thiodicarb + Dipel ES	2.921 ± 0.036a	3.94 ± 0.15a	27.6 ± 0.67a

Means within columns followed by a common letter are not significantly different ($P > 0.05$, Duncan's [1955] multiple range test).

Discussion

Cotton varieties possessing resistance to tobacco budworm and bollworm integrated with reduced frequency or rates of chemical control agents can be effective in managing these pests. Although no cotton cultivar/line and insecticide treatment interaction was observed in 1988 for those variables measured, La. HG-660 treated with thiodicarb generally produced results comparable to those for the standard treatment, DPL 41 combined with lambda-cyhalothrin. Because tobacco budworm and bollworm pressure was relatively low in 1989, treatment combinations had no significant effect on these pests or cotton yields.

The results of the 1988 trial indicated La. HG-660 reduced insect infestation and damage, matured significantly earlier and produced yields comparable to those for DPL 41. Lint turnout and boll weight was lower in the La. HG-660 line than DPL 41. The numerical changes in the yields for the two cultivars were related to the lint turnout and boll weight of each cotton cultivar/line. Previous studies with La. HG-660 (1984-1986) and La. FNHG-850075 cotton strains have demonstrated significant reductions in larval damaged fruit compared to that of a susceptible cultivar (Jones et al. 1987, 1988, 1989). Results of those earlier studies also showed that boll weights of La. HG-660 were lower than

those of a commercial cultivar 'Stoneville 213'. However, lint yields and lint turnout La. HG-660 and La. FNHG-850075 were found to be similar to that another commercial standard, DPL 41 (Jones et al. 1987, 1988, 1989).

In 1989, Plots planted to La. FNHG-850075 matured significantly later than plots planted to La. HG-660 and later than plots planted to DPL 41. Previous studies have not shown any differences in maturity between La. FNHG-850075 and that of commercial cultivars (J. E. Jones, unpublished data). Plots planted to all cotton cultivar/lines initiated fruiting within comparable time intervals and followed similar patterns of fruiting until late June. During late June and early July, plots planted to La. FNHG-850075 began to shed high numbers of squares relative to the plots planted to the other cottons. The frego-bract trait of La. FNHG-850075 confers hypersensitivity to the tarnished plant bug, Lygus lineolaris (Palisot de Beauvois) and cotton fleahopper, Pseudatomoscelis seriatus (Reuter), which were present in the plots in low numbers during June (Jones 1972). These pests could be associated with square shed in plots planted to La. FNHG-850075, but adverse weather conditions during that time period may also have contributed to the problem. An extended period of low light intensity from cloudy and rainy weather occurred during June and July coinciding with

square loss in the La. FNHG-850075 plots. Periods of low light intensity have been associated with square abscission in cotton (Christiansen 1986) and fruit retention for FNHG-850075 may be more sensitive to changes in light intensity than for other cottons.

The insecticide treatments used in 1988, thiodicarb and lambda-cyhalothrin, provided tobacco budworm and bollworm control and yields superior to that observed in the control plots. Lambda-cyhalothrin, a larvicide, significantly reduced the number of larval damaged fruit which resulted in increased seed cotton and lint yields compared to that in the thiodicarb treated plots. The lambda-cyhalothrin treatment should be much more effective based on its residual activity against larvae. The rate of the thiodicarb being used (0.28 kg [AI]/ha), is less than the labeled larvicidal rates (0.7-1.0 kg [AI]/ha) and most of the activity against tobacco budworm and bollworm would be ovicidal. This treatment would be ineffective against an established infestation, but thiodicarb at 0.28 kg [AI]/ha has been effective against larvae just after eclosion from the egg (Leonard et al. 1988a). Lambda-cyhalothrin and thiodicarb are likely to produce similar results for the number of larvae in terminals and larval damaged terminals within 2-3 days after insecticide applications. To maximize control by the thiodicarb treatment, more intensive

scouting procedures are required to improve application timing or the number of treatments needs to be increased.

The boll weevil was not a factor influencing yield in the 1988 test. Multiple applications of organophosphates were used to maintain populations below damaging levels and no significant differences in boll weevil damaged fruit between the cultivar/lines or among the insecticide treatments were observed. In 1989, boll weevil infestations were higher and more difficult to control. Although organophosphates were used to manage this pest, there were significant differences in boll weevil damaged fruit among treatments. The plots planted to La. FNHG-850075 (treated and untreated) had fewer boll weevil damaged fruit compared to numbers in the untreated DPL 41 and untreated La. HG-660 plots. These differences were associated with the frego bract trait expressed by La. FNHG-850075 that confers resistance to boll weevils through antixenosis. Also, the frego bract trait has been associated with improved application efficiency by allowing better coverage of the flower bud with insecticides.

Failures to control soybean looper in cotton and soybean with the pyrethroids has been attributed in part to insecticide selection of adults in cotton fields (Leonard et al. 1990). The lowest IR was observed in the plots treated with lambda-cyhalothrin but considerable numbers of

larvae survived in spite of the insecticide treatments in the plots and likely contributed to the next field generation in soybean. These data indicate pyrethroid selection pressure is also being unintentionally exerted upon soybean looper larvae feeding in cotton.

The resistance mechanisms in La. HG-660 and La. FNHG-850075 advanced lines are associated with a high density of glands containing gossypol-type terpenoid compounds on the calyx lobes of the flower bud. An increase in gossypol in the flower bud generally corresponds to higher levels of this terpenoid in the seed. Although increased levels of seed gossypol are undesirable, advanced lines with other allelochemicals are being constantly evaluated and agronomic properties are being improved. These cotton strains also possess glabrous stems that can reduce tobacco budworm and bollworm oviposition by providing less preferred surfaces for egg deposition on the plant. Previous studies have associated this glabrous trait with a reduction in the number of eggs and small larvae (Lukefahr et al. 1971, 1975). As mentioned earlier, La. FNHG-850075 also expresses the frego bract trait that improves application efficiency and imparts some resistance to the boll weevil.

Currently, pyrethroid resistance has been documented in tobacco budworm populations across the cotton belt (Leonard et al. 1988b). Alternative tobacco budworm and

bollworm larvicides are more expensive and are generally not as effective as the pyrethroids against the boll weevil. Resistance management plans are being implemented in a concerted effort to maintain the efficacy of the pyrethroids (Graves et al. 1988), but such strategies are not likely to prevent the ultimate loss of these compounds. Therefore, management strategies not involving the pyrethroids should be developed as rapidly as possible as a long term solution. This study suggests the use of cotton varieties possessing resistance to tobacco budworm and bollworm in combination with insecticides as an alternative scheme of management, but further research must be conducted to determine specific cotton line and insecticide treatment interactions for an optimum combination.

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CHAPTER III

OVICIDAL EFFECTS OF SELECTED INSECTICIDES AGAINST PYRETHROID-RESISTANT AND -SUSCEPTIBLE TOBACCO BUDWORM (LEPIDOPTERA: NOCTUIDAE)

Introduction

Pyrethroid resistance in field strains of tobacco budworm, Heliothis virescens (F.), has been reported in most of the cotton producing states in which this insect is a pest of cotton (Leonard et al. 1988a, Luttrell et al. 1987, Plapp et al. 1987). Management strategies have been developed to delay the widespread development of resistance to levels that will prohibit the use of the pyrethroids (Anonymous 1986, Plapp 1987, Graves et al. 1988). These strategies commonly recommend the use of an ovicide in combination with a larvicide and the timing of insecticide applications against infestations of tobacco budworm in the egg or neonate larval stages of development. Chlordimeform was an important component in mixtures with pyrethroids and provided both synergistic and ovicidal activity (Plapp 1976, 1979; Campanhola & Plapp 1987; Graves et al. 1988). The loss of chlordimeform during 1989 has caused researchers to intensify efforts in the evaluation of chlordimeform alternatives as ovicides and synergists.

Numerous studies have evaluated the ovicidal activity of a variety of selected organochlorine, organophosphate, carbamate, formamidine and pyrethroid insecticides (Walker 1966, Tysowsky & Gallo 1977, Bull & House 1978, Campbell et al. 1979, Pitts & Pieters 1980, Horowitz et al. 1987,

Bradley & Agnello 1988) as well as insect growth regulators (Masner et al. 1987) to eggs of tobacco budworm and bollworm, Helicoverpa zea (Boddie). Because tobacco budworm eggs may be present on cotton plants throughout much of the growing season, they may be exposed to a variety of insecticides that are applied for control of other cotton pests. These eggs are inadvertently under insecticide selection pressure that can lead to altered levels of susceptibility. In some of these studies, differences in the insecticide susceptibility of eggs from different tobacco budworm strains have been observed. Bull & House (1978) reported that chlordimeform was less toxic to eggs of a methyl parathion-resistant strain of tobacco budworm than to eggs of a -susceptible strain. The toxicity of chlordimeform, amitraz, methomyl, monocrotophos, acephate, methyl parathion and fenvalerate also were observed to be significantly less toxic to eggs from a field strain compared to eggs from a laboratory strain (Horowitz et al. 1987).

The relative insecticidal susceptibility of eggs from different tobacco budworm strains is not well known because standard monitoring procedures for insecticide resistance typically focus on the larval or adult stages of this insect. Current management strategies for tobacco budworm target insecticide applications to the egg and early larval

stages. These practices coupled with the potential for resistance development in this pest justify determining the susceptibility of eggs from pyrethroid-resistant tobacco budworms to selected insecticides. The objective of this study was to evaluate the ovicidal toxicity of insecticides from several classes against strains of tobacco budworm with varying levels of pyrethroid susceptibility in the larval stage.

Methods and Materials

Insects

Two laboratory strains and one field strain (FIELD-89) were included in this study. The pyrethroid-susceptible laboratory strain, PY-S, was initiated from a collection of larvae obtained from a cotton field in Louisiana during 1977. This collection was made before widespread use of the pyrethroids in cotton and has been maintained in continuous culture in the laboratory without exposure to insecticides. The pyrethroid-resistant laboratory strain, PY-R, was obtained from ICI Americas Inc. (Wilmington, Del.) during September 1988. This strain had been continuously pressured with cypermethrin but was not further exposed to insecticides after being colonized in our laboratory. The egg bioassays were conducted during the F_5-F_6 generations of the PY-R strain in our laboratory. The FIELD-89 strain was established from a collection of ca. 380 eggs taken from a cotton field on the Northeast Research Station, St. Joseph, La., on 24 and 25 August 1989. This strain represents the survivors of an insecticide trial to evaluate the ovicidal efficacy of selected pyrethroids. No other insecticide had been applied to this field, but the test site was in close proximity to several large fields of commercially produced cotton that received multiple

applications of pyrethroids, organophosphates and carbamates throughout the season.

The tobacco budworm strains were all reared in a similar manner according to the laboratory procedures described by Leonard et al. (1988a). Larvae were fed a modified pinto bean and wheat germ artificial diet (Shour & Sparks 1981). Adults were held in 3.78 L cardboard cartons covered with cotton gauze and were fed an aqueous (1:10 sugar:water ratio) solution. Tobacco budworm adults oviposited on the cotton gauze and these eggs were used in the egg bioassays or placed on artificial diet for larval rearing. Insects were held at a 14:10 (L:D) photoperiod, 28 $\pm$ 3° C and 65-70% RH for the duration of this experiment.

Insecticides

Technical grade insecticides were used in the bioassays to determine pyrethroid toxicity to larvae, but formulated materials were used in the tests to compare ovicidal activity. Samples of cypermethrin (FMC Corp., Middleport, N.Y.), lambda-cyhalothrin (ICI Americas Inc., Wilmington, Del.) and esfenvalerate (E. I. DuPont de Nemours & Co. Inc., Wilmington, Del.) were obtained from the manufacturers. Technical materials were diluted with acetone to a 10% stock solution and refrigerated at 15°C until needed for bioassays. The formulated insecticides evaluated for ovicidal activity were esfenvalerate (60 g/liter

emulsifiable concentrate [EC]; E. I. DuPont de Nemours & Co. Inc., Wilmington, Del.), lambda-cyhalothrin (120 g/liter EC; ICI Americas Inc., Wilmington, Del.), thiodicarb (384 g/liter flowable liquid [F]; Rhone-Poulenc, Research Triangle Park, N.C.), methomyl (288 g/liter liquid [L];E. I. DuPont de Nemours & Co. Inc., Wilmington, Del.), amitraz (180 g/liter EC; NOR-AM Chemical Co., Wilmington, Del.), chlordimeform (480 g/liter EC; CIBA-GEIGY Corp., Greensboro, N.C.) and profenofos (960 g/liter EC; CIBA-GEIGY Corp., Greensboro, N.C.). The numbered compound SN 49844 (BTS-27712, N'-[2,4-dimethylphenyl]-N-methylformamidine, 100% soluble powder [SP]), which was obtained from NOR-AM Chemical Co., Wilmington, Del. was also evaluated for ovicidal activity.

Bioassays

Tests to determine pyrethroid susceptibility in tobacco budworm larvae were done according to a standard method for determining insecticide resistance in tobacco budworm and bollworm (Anonymous 1970). Ten to fifteen larvae (third stage, mean weight = 25 $\pm$ 4 mg) were treated with each of four to six concentrations of insecticide in acetone. Each dose was replicated at least three times. One microliter of the insecticide solution or acetone (controls) was applied to the dorsal surface of the thorax of each larva. Treated insects were individually held in 29.7 ml plastic

cups containing artificial diet. Mortality data were recorded 72 h after treatment. A larva was considered dead if it failed to upright itself after being overturned with a blunt probe. Dose-mortality regressions were determined with a microcomputer based probit analysis (MicroProbit 3.0, T. C. Sparks & A. P. Sparks, unpublished, Greenfield, Ind.). All data were corrected for mortality (< 5%) in the controls (Abbott 1925). Significant differences among LD_{50} values were determined by the failure of 95% confidence limits (CL) to overlap. The PY-R strain was tested with pyrethroids one generation before the ovicidal evaluation. The FIELD-89 strain was bioassayed with cypermethrin and esfenvalerate in the F_1 generation and with lambda-cyhalothrin during the F_2 generation.

Insecticide toxicity to eggs was evaluated using a dipping technique modified from previous methods (Horowitz et al. 1987). Sheets of cotton gauze covering the adult oviposition chambers were removed daily, placed in inflated plastic bags and held for 24 h to allow further egg development. Pieces of cotton gauze were cut to contain a sample of 15 to 30 fertile eggs. Fertility (embryonic development) was indicated by the presence of a red band (germ band) around the circumference of the eggs. These samples were submerged into an insecticide distilled water (pH = 6.4 - 6.8) solution for 4 s and air dried for 30 min

in a fume hood. After drying, the treated egg samples were placed on No. 1 filter paper in plastic petri dishes (10 cm). Four to seven doses in three replications were used to obtain a dose-mortality line for each insecticide. Egg mortality was determined at 72 h after treatment. Any larva that fully eclosed from the chorion was recorded as a hatched egg. All data were analyzed with the same procedures used in the larval tests. Control mortality in the egg bioassays ranged from 0 to 12%.

Results

Larval LD_{50}'s for the PY-R strain to cypermethrin, esfenvalerate and lambda-cyhalothrin were 207, 239 and 65 times higher, respectively, than LD_{50}'s for the PY-S strain (Table 1). The larval LD_{50}'s for cypermethrin and esfenvalerate on the FIELD-89 strain were much lower than those of the PY-R strain being only 3.1 and 3.0 times, respectively, higher than those reported for the PY-S strain. There was no significant difference in the toxicity of lambda-cyhalothrin between the PY-S and FIELD-89 strains.

Esfenvalerate and lambda-cyhalothrin were significantly more toxic to eggs of the PY-S strain than other insecticides except for chlordimeform and SN 49844 (Table 2). Based on LC_{50} values, chlordimeform was one of the most toxic insecticides to eggs of the three tobacco budworm strains, while profenofos was consistently the least toxic. All insecticides except profenofos were more toxic to eggs of the PY-S strain compared to the toxicity of the same insecticide to eggs of the PY-R strain. All insecticides except for profenofos and methomyl were more toxic to eggs of the PY-S strain when compared to their toxicity to eggs of the FIELD-89 strain. The highest level of resistance was found with esfenvalerate (13.6 times) and lambda-cyhalothrin (34.9 times) in eggs of the PY-R strain. Relatively low

levels of resistance were found to all other insecticides except chlordimeform (5.4 times) in this strain. The highest level of resistance in the FIELD-89 strain was to lambda-cyhalothrin (11.7 times). This strain also exhibited a moderate level of resistance to chlordimeform (6.5 times) and SN 49844 (7.2 times). There was no significant difference among LC_{50}'s of the tobacco budworm strains to profenofos.

Table 1. Toxicity of selected pyrethroids to larvae of pyrethroid-susceptible (PY-S), pyrethroid-resistant (PY-R) and field (FIELD-89) strains of tobacco budworm

Insecticide	Strain[a]	No. Tested	Slope + SE	LD_{50}[b]	(95% CL)	RR[c]
Cypermethrin	PY-S	150	3.50 ± 0.60	1.61	(1.32-1.93)	---
	PY-R	237	1.89 ± 0.26	333.89	(268->400)	207.4
	FIELD-89	221	1.87 ± 0.24	4.96	(3.92-6.46)	3.1
Esfenvalerate	PY-S	150	1.74 ± 0.38	0.42	(0.11-0.49)	---
	PY-R	142	1.43 ± 0.28	100.4	(61.08->145)	239.0
	FIELD-89	294	1.73 ± 0.17	1.25	(1.00-1.58)	3.0
Lambda-cyhalothrin	PY-S	150	2.64 ± 0.37	0.93	(0.68-1.14)	---
	PY-R	178	1.34 ± 0.22	60.42	(33.09-89.08)	65.0
	FIELD-89	190	1.49 ± 0.21	1.17	(0.84-1.69)	1.3

[a] Results for LSU PY-S strain adapted from Leonard et al. (1988b).

[b] Dosages are reported in μgrams of insecticide per g of larval weight.

[c] RR (resistance ratio) = (LD_{50} PY-R or FIELD-89 strain)/(LD_{50} PY-S strain).

Table 2. Toxicity of insecticides to eggs of pyrethroid-susceptible (PY-S), pyrethroid-resistant (PY-R) and field (FIELD-89) strains of tobacco budworm

	PY-S Strain			PY-R Strain				FIELD-89 Strain			
Insecticide	n	Slope ± SE	LC_{50}(mg[AI]/ml) (95% CL)	n	Slope ± SE	LC_{50}(mg[AI]/ml) (95% CL)	RR[a]	n	Slope ± SE	LC_{50}(mg[AI]/ml) (95% CL)	RR[a]
Pyrethroids											
l-Cyhalothrin	527	1.95 ± 0.18	0.017 (0.014-0.019)	413	1.34 ± 0.14	0.593 (0.467-0.774)	34.9	206	1.98 ± 0.27	0.199 (0.063-0.358)	11.7
Esfenvalerate	439	2.12 ± 0.15	0.043 (0.036-0.050)	368	1.31 ± 0.15	0.586 (0.446-0.821)	13.6	NB[b]			
Carbamates											
Methomyl	302	1.77 ± 0.20	0.081 (0.063-0.103)	350	1.92 ± 0.18	0.157 (0.130-0.192)	1.9	332	2.15 ± 0.37	0.108 (0.09-0.13)	1.3
Thiodicarb	1173	2.49 ± 0.14	0.218 (0.198-0.241)	712	2.41 ± 0.19	0.287 (0.256-0.323)	1.3	586	2.63 ± 0.16	0.851 (0.596-1.406)	3.9
Formamidines											
Chlordimeform	792	1.79 ± 0.14	0.016 (0.014-0.020)	839	1.03 ± 0.10	0.086 (0.069-0.106)	5.4	308	2.17 ± 0.22	0.104 (0.088-0.124)	6.5
SN 49844	542	1.34 ± 0.11	0.043 (0.033-0.054)	473	1.03 ± 0.12	0.125 (0.096-0.166)	2.9	313	1.87 ± 0.19	0.309 (0.251-0.379)	7.2
Amitraz	635	1.88 ± 0.20	0.122 (0.098-0.145)	959	2.92 ± 0.16	0.331 (0.277-0.403)	2.7	329	4.61 ± 0.55	0.367 (0.289-0.463)	3.0
Organophosphate											
Profenofos	495	1.48 ± 0.16	1.087 (0.858-1.479)	350	1.3 ± 0.16	1.099 (0.825-1.590)	1.0	388	1.82 ± 0.17	1.182 (0.79-1.64)	1.1

[a] RR (resistance ratio) = (LC_{50} PY-R or FIELD-89 strain)/(LC_{50} PY-S strain).

[b] Not bioassayed.

Discussion

Although different procedures were used to measure pyrethroid susceptibility in eggs and third instar larvae, both techniques detected a significant decrease in pyrethroid toxicity to the PY-R and FIELD-89 strains compared to the PY-S strain. The variation in the susceptibility of eggs from the different tobacco budworm strains to esfenvalerate and lambda-cyhalothrin is probably the result of differential selection pressure with the pyrethroids. The level of resistance to lambda-cyhalothrin in third instar larvae (65.0-fold) and eggs (34.9-fold) was much higher than the resistance level found in larvae (1.3-fold) and eggs (11.7-fold) of the FIELD-89 strain. Although the PY-R strain has only been exposed to cypermethrin as a selecting agent, resistance to esfenvalerate and lamba-cyhalothrin was observed in larvae and eggs of this strain. In a previous study, larvae of a pyrethroid-resistant (labeled ICI) strain from the same source as the PY-R strain were found to be resistant to all pyrethroids to which they were exposed (Campanhola & Plapp 1987).

The toxicities of most of the non-pyrethroid insecticides against the PY-S strain were similar to that obtained against eggs of the PY-R and FIELD-89 strains. The FIELD-89 strain had been exposed to a variety of

insecticides before its removal from the field and was generally less susceptible than the PY-S strain to most of the insecticides tested in this study. Using a similar technique, Horowitz et al. (1987) found significant differences in the LC_{50}'s of pyrethroids, formamidines, carbamates and organophosphates between a field tobacco budworm strain collected in Southern California and a laboratory tobacco budworm strain. Using an insecticide coated vial test, Bagwell & Plapp (1988) found chlordimeform to be significantly more toxic than amitraz to pyrethroid-resistant and -susceptible laboratory tobacco budworm strains. LC_{50} values for eggs of the pyrethroid-resistant strain were determined to be 3.9 and 3.3 times higher for chlordimeform and amitraz, respectively, compared to eggs of the pyrethroid-susceptible tobacco budworm strain. Eggs of a methyl parathion-resistant tobacco budworm strain were less susceptible (2.2-fold) to chlordimeform than eggs of a methyl parathion-susceptible laboratory tobacco budworm strain (Bull & House 1978). The results of this and other similar studies indicate that while eggs of field-collected or insecticide-pressured tobacco budworm strains are less susceptible to insecticides compared to eggs of insecticide-susceptible tobacco budworm strains, the variation in insecticide susceptibility among tobacco budworm strains is very often quite low.

The relative toxicity of different insecticides to tobacco budworm eggs in the present study indicated chlordimeform was the most toxic formamidine and methomyl was the most toxic carbamate tested on the PY-S strain. Other studies (Pitts & Pieters 1980, Gonzales & Allen 1985, Horowitz et. al. 1987, Bradley & Agnello 1988) have also found chlordimeform, methomyl and thiodicarb to be highly toxic to tobacco budworm and bollworm eggs. In our study, amitraz was significantly less active than chlordimeform against tobacco budworm eggs of all strains. Pitts & Pieters (1980) found amitraz to be significantly less toxic than chlordimeform to tobacco budworm eggs, but several field studies have shown that the ovicidal activity of amitraz is equal to that of chlordimeform (Elzen 1989, Leonard et al. 1989, Micinski et al. 1989). A comparison of the toxicity of selected formamidines to tobacco budworm eggs in a laboratory test indicated no significant difference between SN 49844 and chlordimeform, but found chlordimeform to be slightly more toxic to bollworm eggs (Gemrich et al. 1976). The pyrethroids, esfenvalerate and lambda-cyhalothrin, were highly active ovicides against the PY-S and FIELD-89 strains in this study. Several studies have demonstrated the toxicity of various pyrethroids to tobacco budworm eggs (Pitts & Pieters 1980, Jany 1984, Horowitz et al. 1987, Leonard et al. 1989). Profenofos

exhibited the lowest ovicidal activity in this study, but field tests have indicated that the contact toxicity of profenofos to tobacco budworm eggs was not significantly different from that of chlordimeform and thiodicarb (Leonard et al. 1989, Micinski et al. 1989). The particular assay used in this test may have influenced the activity of this compound.

The mode of insecticidal action in insect eggs is not well understood and at least two types of mortality are associated with death of the developing larva. The embryo in the egg may be killed or the developing larva dies as it feeds on the chorion during eclosion. Both types of mortality were observed in this study, but no attempt was made to differentiate between the two. These variations in embryo death have been observed in studies that have evaluated the ovicidal toxicity of several different classes of insecticides including formamidines, carbamates and organophosphates (Walker 1966, Gemrich et al. 1976, Bradley & Agnello 1988). Studies with chlordimeform treated Spodoptera littoralis (Boisduval) eggs have shown that larval survival can be increased if the larva is removed from the chorion before it begins to feed during eclosion (Salvisberg et al. 1980). For insecticide treated ova, the ingestion of egg materials during the hatching process contributes significantly to intoxication of the developing

embryo. In our experiment the eggs were completely submerged in the insecticide-water solution and all of the chorion should have been exposed to the insecticide. Although insecticides are taken up by the eggs at the time of treatment, they may not become lethal until further development depending on the dose that actually reaches the embryo. They may be retained in the egg until further embryonic development or the dose of insecticide becomes lethal through continuous exposure and/or metabolism. Storage either within the egg or on the chorion would increase the potency of the toxicant by increasing the overall amount received by the embryo. Although the structural characteristics of the chorion are designed to prevent desiccation, there are openings in the chorion (aeropyles and micropyle) for gas exchange to occur (Smith & Salkeld 1966). Insect eggs have an extensive respiratory system, that may allow uptake and storage of a toxicant without affecting the embryo until a later stage of development.

As the embryo undergoes significant changes in physiology from an inactive physical stage to one that is capable of movement, stress levels are increased. The formamidines at sublethal doses are capable of disrupting behavioral patterns in insects, thereby increasing stress and accentuating the potential for starvation and

dehydration (Lund et al. 1979).

Insecticides used against tobacco budworm and bollworm in cotton generally target the larval stages. The recommended use rates are higher than the amount that is be lethal to the egg stage. In a field study in Australia, researchers found no significant differences in egg or early instar larval (< 3 d old) mortality between pyrethroid-resistant and -susceptible Heliothis armigera (Hubner) when eggs were exposed to fenvalerate (Daly et al. 1988). In this case, the field dose may actually be an overdose of insecticide for this developmental stage because resistance was observed in older larvae (> 4 d old). However, in other studies, lambda-cyhalothrin at recommended field rates was less toxic to eggs and neonate larvae of a pyrethroid-resistant tobacco budworm strain compared to eggs and neonate larvae of a -susceptible strain (Treacy et al. 1988, Hopkins et al. 1989). Thus, pyrethroid resistance in eggs may be detected in laboratory bioassays but may not be recognized in the field if recommended rates of insecticides are being used. The ovicidal activity of insecticides to tobacco budworm should not be underestimated as pyrethroid resistance management strategies emphasize the use of ovicides and application timing to coincide with the egg or early larval stages.

Consideration should be given to a standardized system

of monitoring insecticide susceptibility in the egg stage of tobacco budworm. A monitoring system is fundamental to extending the usefulness of insecticides and to allow appropriate measures to be taken before such a problem would become unmanageable.

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CHAPTER IV

ALTERNATIVE OVICIDES TO CHLORDIMEFORM FOR CONTROL OF TOBACCO BUDWORM AND BOLLWORM (LEPIDOPTERA: NOCTUIDAE) IN COTTON

Introduction

Insecticides are a major component of tobacco budworm, Heliothis virescens (F.) and bollworm, Helicoverpa zea (Boddie), management strategies in cotton, Gossypium hirsutum L., production in the mid-south. Tobacco budworm and bollworm infestations often coincide to reach extremely high levels in cotton fields, and insecticide mixtures that possess both larvicidal and ovicidal activity are required for effective control. Synergistic interactions of insecticide mixtures, particularly involving chlordimeform, have been indicated to be effective against tobacco budworm larvae (Plapp 1976, 1979) and eggs (Horowitz et al. 1987, Bagwell & Plapp 1988). The recent development of pyrethroid resistance in tobacco budworm has increased emphasis on the use of insecticides with ovicidal and synergistic properties in combination with pyrethroids. Such mixtures have already been recommended in pyrethroid resistance management strategies for controlling tobacco budworms and delaying widespread resistance (Anonymous 1986, Plapp 1987, Graves et al. 1988).

Chlordimeform and methomyl have been the ovicides commonly used against eggs of tobacco budworm and bollworm in cotton (Pitts & Pieters 1980). However, the Environmental Protection Agency banned chlordimeform use on

cotton in the United States after September 1989. Methomyl is highly toxic to these pests initially but has relatively low residual activity compared to chlordimeform (Bradley & Agnello 1988). Recently, several studies have confirmed the ovicidal properties of thiodicarb, and it has been used extensively during 1988 and 1989 (Gonzales & Allen 1985, Bradley & Agnello 1987).

Several studies have indicated that selected organophosphates, carbamates, formamidines, pyrethoids and insect growth regulators possess ovicidal properties against tobacco budworm or bollworm in the laboratory (Horowitz et al. 1987, Masner et al. 1987, Bagwell and Plapp 1988) and in the field (Pitts & Pieters 1980, DuRant & Moore 1989, Hopkins et al. 1989). However, most of these studies were concerned only with establishing the presence of ovicidal activity and did not attempt to determine the effective field rates relative to other insecticides. Furthermore, the effects on newly hatched larvae from insecticide treated eggs or from exposure to treated foliage was not examined.

The primary objective of this study was to determine the contact and residual ovicidal properties of insecticides from several classes against tobacco budworm and bollworm. A second objective was to measure residual toxicity of these insecticides to newly hatched larvae of field strains.

Methods and Materials

Insecticides

The toxicity of 23 insecticides at multiple use rates to eggs and neonate larvae were determined in field tests during 1987-1989. The materials tested were methomyl (216 g/liter liquid [L] or 288 g/liter L; E. I. DuPont de Nemours & Co., Wilmington, Del.), thiodicarb (384 g/liter flowable [F]; Rhone-Poulenc, Research Triangle Park, N.C.), oxamyl (240 g/liter L; E. I. DuPont de Nemours & Co., Wilmington, Del.), fenoxycarb (240 g/liter emulsifiable concentrate [EC]; Abbott Laboratories, North Chicago, Ill.), amitraz (180 g/liter EC; NOR-AM Chemical Co., Wilmington, Del.), chlordimeform (480 g/liter EC; CIBA-GEIGY Corp., Greensboro, N.C.), SN 49844 (100% soluble powder [SP], N'-(2,4-dimethylphenyl)-N-methylformamidine; NOR-AM Chemical Co., Wilmington, Del.), endosulfan (360 g/liter EC; FMC Corp., Philadelphia, Penn.), acephate (75% and 90% SP; Valent U.S.A. Corp., Walnut Creek, Calif.), azinphosmethyl (240 g/liter EC; Mobay Corp., Kansas City, Mo.), malathion (492 g/liter EC; American Cyanamid Corp., Princeton, N.J.), methamidophos (480 g/liter EC; Mobay Corp., Kansas City, Mo.), profenofos (720 and 960 g/liter EC; CIBA-GEIGY Corp., Greensboro, N.C.), sulprofos (720 g/liter EC; Mobay Corp., Kansas City, Mo.), bifenthrin (240 g/liter EC; FMC

Corporation, Philadelphia, Penn.), cyfluthrin (240 g/liter EC; Mobay Corp., Kansas City, Mo.), cypermethrin (360 g/liter EC; ICI Americas Inc., Wilmington, Del.), esfenvalerate (60 and 79 g/liter EC; E. I. DuPont de Nemours & Co., Wilmington, Del.), fenvalerate (288 g/liter EC; E. I. DuPont de Nemours & Co., Wilmington, Del.), fluvalinate (240 g/liter F; Zoecon Corp., Palo Alto, Calif.), lambda-cyhalothrin (120 g/liter EC; ICI Americas Inc., Wilmington, Del.), permethrin (240 g/liter EC; ICI Americas Inc., Wilmington, Del.) and tralomethrin (36 and 108 g/liter EC; Hoechst-Roussel Agri-Vet Co., Somerville, N.J.).

Field Tests

Field tests were conducted during 1987 at the Northeast Research Station near St. Joseph, La. (SJ-87). In 1988, tests were done at the Dean Lee Research Station, Alexandria, La. (DL-88), Red River Research Station, Bossier City, La. (RR-88), in a commercial cotton field near St. Joseph, La. (SJ-88A) and at the Northeast Research Station (SJ-88B). Tests were done during 1989 at the Red River Research Station (RR-89), the Northeast Research Station (SJ-89) and at the Macon Ridge Branch of the Northeast Research Station, Winnsboro, La. (MR-89). The test plots had not been treated with foliar insecticides, but aldicarb + terraclor + terrazole (0.56 + 1.12 + 0.28 kg [AI]/ha) was applied at planting to control early season pests in all

tests.

The SJ-87 trial was done on cotton, 'Deltapine 41' planted 8 April on 1.0 m centers and divided into plots measuring 4 rows by 27.3 m. Treatments were arranged in a randomized complete design and replicated four times. Insecticides were applied on 18 August with a handheld CO_2 charged sprayer with two TX-6 hollow cone nozzles per row calibrated to deliver a total volume of 56.8 liters/ha at 2.9 kg/cm^2. No rainfall occurred during this test.

The DL-88 trial was done on an experimental cotton (LA Barb 392-FN) planted 15 June on 1.0 m centers and divided into plots measuring 4 rows by 22.5 m. Treatments were arranged in a randomized complete block design and replicated four times. Insecticides were applied on 15 August with a handheld CO_2 charged sprayer with two TX-6 hollow cone nozzles (Spraying Systems, Inc.) per row calibrated to deliver a total volume of 56.8 liters/ha at 2.9 kg/cm^2. No rainfall occurred during this test.

The RR-88 and RR-89 tests were conducted on plots of cotton, 'Deltapine 50', both of which were planted 25 April on 1.0 m centers. Plots measured 4 rows by 30.5 m in the RR-88 trial and 8 rows (only 4 were treated) by 30.5 m in the RR-89 trial. Treatments were arranged in a randomized complete block design with four replicates in both tests. Insecticides were applied 16 June (RR-88) and 21 June (RR-

89) with a high-clearance plot sprayer equipped with a CO_2 system and two TX-3 hollow cone nozzles per row calibrated to deliver a total volume of 28.5 liters/ha at 4.2 kg/cm^2. No rainfall was recorded during the RR-88 trial, but a trace amount of rain (< 0.2 cm) was recorded on 21 June, 1989 following the 4 h sample of the RR-89 trial.

The SJ-88A test was conducted in a field of commercial cotton, 'Deltapine 20', planted 9 April. The SJ-88B test was done in plots of cotton, 'Deltapine 90', planted 24 May. The SJ-89 test was done in a field of cotton, 'Deltapine 20', planted 15 June. Plots in these tests consisted of four treated rows on 1.0 m centers and 30.3 m, 15.2 m and 15.2 m long for the SJ-88A, SJ-88B and SJ-89 tests, respectively. Treatments were arranged in a randomized complete block design and replicated four times. Insecticides were applied on 9 June (SJ-88A) and 21 July (SJ-88B) with a small tractor equipped with a compressed-air system and two TX-6 hollow-cone nozzles per row calibrated to deliver a total volume of 47.3 liters/ha at 2.5 kg/cm^2. In the SJ-89 test, insecticides were applied on 23 August with a high clearance plot sprayer equipped with a CO_2 charged application system and two TX-6 hollow cone nozzles per row calibrated to deliver a total volume of 47.3 liters/ha at 2.5 kg/cm^2. No rainfall was recorded during the SJ-88A test. A trace amount of rainfall was recorded

24 h after treatment in the SJ-89 trial.

The MR-89 test was done in a field of cotton, 'Stoneville 825', planted 23 June on 1.0 m centers. Plots consisted of 4 rows by 15.0 m. Treatments were arranged in a randomized complete block design and replicated 3 times. Insecticides were applied on 29 August with a high clearance plot sprayer equipped with a CO_2 charged application system and two TX-3 hollow cone nozzles per row calibrated to deliver a total volume of 23.6 liters/ha at 3.1 kg/cm^2. A trace amount of rainfall was recorded on 29 August following the 2 h sample.

These tests were conducted on cotton plots naturally infested with tobacco budworm and bollworm eggs. During all tests, eggs were randomly collected from the upper one third of the plant canopy on the two center rows of each plot. Only those eggs less than 24 h old with a milky white color and no obvious embryonic development (darkening of the egg contents or formation of a red band around the chorion circumference) were collected from the treated plots in all tests except SJ-87. In the SJ-87 test, egg collections consisted primarily (> 85%) of freshly oviposited eggs, but due to low egg density and to keep a consistent sample size some eggs more than 1 d old were collected. Samples of eggs (10/treated plot) were taken from the plots 4-6 h and 48 h posttreatment to measure contact and residual toxicity in

all tests except SJ-87 (48 h sample consisted of 5 eggs/plot). Additional samples of eggs were collected at 24 and 96 h posttreatment in the SJ-88A test and at 24, 72 and 96 h posttreatment in the MR-89 test. During oviposition these eggs are glued to the plant foliage; therefore, the eggs and the attached cotton foliage were placed in plastic bags and transported to the laboratory. Each egg and a small piece of adjoining plant tissue were placed in a 29.7 ml plastic cup partially filled with artificial diet for rearing tobacco budworm and bollworm (Shour & Sparks 1981). The cups were capped and inverted so that the egg was not in direct contact with the rearing medium. The cups were held at room temperature (23.8-24.7 °C) and mortality to eggs and larvae was measured either at 96 or 120 h after removal from the field. Egg mortality was based on failure of the egg to hatch or incomplete larval eclosion from the chorion. Larval mortality was recorded if the larva had completely emerged from the chorion and was unable to translocate. Survivors of the egg cohort in the untreated control were reared to adults to determine species composition.

Data Analyses

Data were analyzed separately for each test and the results were corrected for mortality in each respective control treatment with Abbott's (1925) formula. These data

were transformed (arcsine square root [n + 0.01]) and subjected to analysis of variance procedures (SAS Institute 1988). Treatment means were compared to the control mean in each trial with Dunnett's one tailed t-test (P = 0.05, Steel & Torrie 1980).

Results

The species composition of the populations observed in the field tests was determined to be predominantly tobacco budworm except in the RR-89 trial (Table 1). The percent of the population found to be tobacco budworm ranged from 19.5% in the RR-89 test to 100% in the SJ-88A and SJ-89 tests. Control mortality for eggs in the 4 h posttreatment sample was extremely variable and ranged from 7.0% in the MR-89 trial to 41.0% in the RR-88 test. Egg mortality in the 48 h sample was generally lower than that observed for the 4 h sample, although in the RR-89 test egg mortality was determined to be 28.3%. There was no larval mortality observed in the 4 and 48 h posttreatment samples from the control treatments of all tests except in the SJ-88B (2.5% larval mortality) and RR-88 (5.2% larval mortality) tests.

Most of the insecticide treatments evaluated in field tests showed high levels of initial toxicity (4 h sample) to eggs but the residual activity (48 h sample) against eggs was generally much lower (Table 2). Egg mortality in the 4 h posttreatment samples was significantly higher for all treatments in one or more field tests except for the methomyl (0.071 kg [AI]/ha, RR-89 trial) and profenofos (0.563 kg [AI]/ha, RR-88 trial) treatments compared to the egg mortality in the control treatment of each respective

test. All rates of thiodicarb, chlordimeform, SN 49844, sulprofos, bifenthrin, cyfluthrin, cypermethrin, esfenvalerate and tralomethrin tested in multiple tests consistently reduced egg hatch below that observed for the respective control treatments in the 4 h posttreatment samples.

In the 48 h posttreatment samples, fewer treatments significantly reduced egg hatch compared to the respective controls (Table 2). The ovicidal performance of methomyl, thiodicarb and fenoxycarb was sporadic, and these compounds demonstrated significant ovicidal activity in only selected tests. However, the formamidines, chlordimeform and SN 49844, consistently increased egg mortality above that in the control treatment. The amitraz (0.281 kg [AI]/ha) treatments significantly reduced egg hatch in the 48 h posttreatment samples compared to the respective control treatments in all field tests except RR-88. None of the organophosphate treatments except profenofos (1.125 kg [AI]/ha, SJ-87 test) significantly influenced egg hatch in 48 h posttreatment samples. Residual ovicidal activity of the pyrethroid treatments was measured only in a few field tests, but bifenthrin, cypermethrin and lambda-cyhalothrin were found to significantly reduce egg hatch compared to their respective controls in the 48 h posttreatment samples.

Although the larvicidal activity of thiodicarb at 4

and 48 h after treatment was inconsistent, this compound significantly increased larval mortality compared with the control at 0.140 and 0.281 kg [AI]/ha. Larval mortality for the formamidines (chlordimeform, amitraz and SN 49844) at 4 and 48 h after treatment were not significantly different from mortality observed in the control. The acephate and sulprofos treatments had significantly higher larval mortality than the control at 4 h after treatment. None of the organophosphate treatments in any of the field tests showed larvicidal activity at 48 h after treatment. The pyrethroids were more active against hatching larvae than the other classes of insecticides tested. All of the pyrethroid treatments except bifenthrin and tralomethrin in the SJ-89 test, cypermethrin in the MR-89 trial and lambda-cyhalothrin in the RR-88 test significantly increased larval mortality compared to their respective control treatments in the 4 h posttreatment sample.

The ovicidal activity of most insecticide treatments did not persist beyond 48 h after treatment (Fig. 1 & 2). In the SJ-88A trial (Fig. 1), only the chlordimeform treatment demonstrated significant ovicidal activity in the 96 h posttreatment sample. In the MR-89 trial (Fig. 2), residual ovicidal activity was greatly reduced 24 h after treatment due to rainfall and only the amitraz and fenoxycarb treatments had significant ovicidal activity

which persisted to 24 h after treatment. None of the treatments in this trial exhibited ovicidal activity beyond 48 h after treatment.

Table 1. Species composition and control mortality of eggs observed in each field experiment

		Species (%) Composition		Control Mortality (%) Egg		Control Mortality (%) Larvae	
Test	N[a]	TBW[b]	BW[b]	4 h	48 h	4 h	48 h
SJ-87	37	68.4	31.6	23.7	17.5	0.0	0.0
SJ-88A	63	100.0	0.0	18.6	7.8	0.0	0.0
SJ-88B	32	93.7	6.3	10.8	---	2.5	---
DL-88	44	52.3	47.7	40.0	5.3	0.0	0.0
RR-88	58	93.2	6.8	41.0	13.1	5.2	0.0
SJ-89	39	100.0	0.0	23.2	5.0	0.0	0.0
MR-89	48	93.7	6.3	7.0	6.6	0.0	0.0
RR-89	41	19.5	80.5	38.5	28.3	0.0	0.0

[a] Sample cohort of eggs from the control treatment surviving to the adult stage

[b] TBW (tobacco budworm); BW (bollworm)

Table 2. Summary of ovicidal activity of selected insecticides and residual toxicity to newly hatched larvae of tobacco budworm and bollworm during 1987-1989

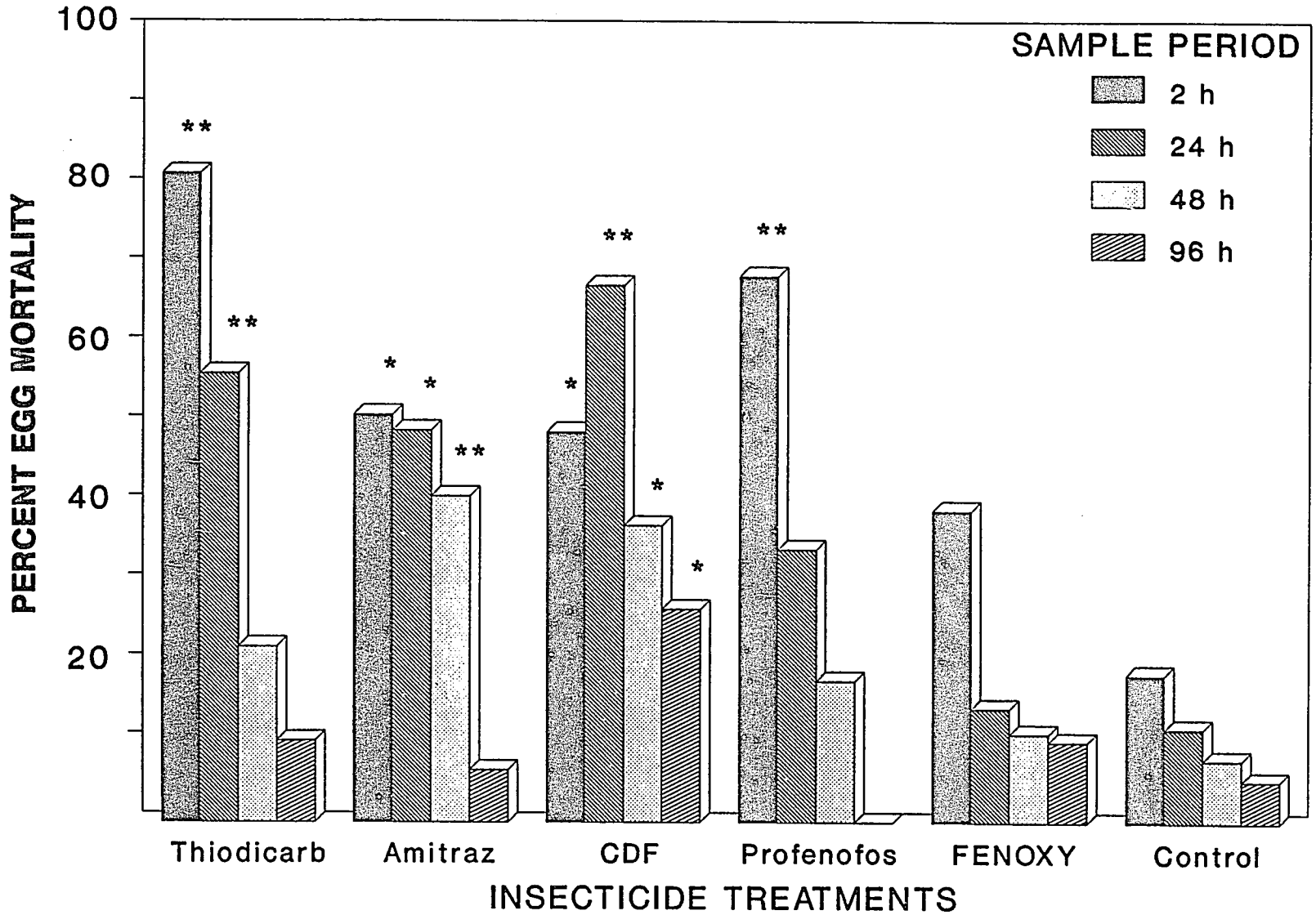
Insecticide	Rate kg [AI]/ha	Test	% Egg Mortality 4-h	% Egg Mortality 48-h	% Larval Mortality 4-h	% Larval Mortality 48-h
Carbamates						
Methomyl	0.071	RR-89	20.2*	11.6	8.3	9.4
	0.140	SJ-87	74.7*	10.7	5.1	0.0
	0.140	SJ-88A	53.7*	20.3	16.8	4.2
	0.140	RR-89	33.6*	5.6*	14.6*	0.0
	0.281	MR-89	76.8*	21.5	30.5*	4.7
Thiodicarb	0.140	SJ-87	64.7*	9.1	24.9*	29.9*
	0.140	SJ-88A	72.4*	20.3*	25.4*	37.7*
	0.140	DL-88	61.3*	36.0	11.8*	7.2
	0.140	SJ-88B	52.6*	---	28.2*	---*
	0.140	RR-89	51.9*	2.3	27.1*	24.3*
	0.214	SJ-88A	77.8*	15.5*	25.2*	31.6*
	0.281	SJ-87	85.7*	60.1*	12.4	12.4
	0.281	MR-89	74.2*	14.4*	8.3	15.8
	0.281	SJ-89	57.4*	14.3*	8.3	12.5*
	0.675	SJ-87	83.6*	17.2	0.0	68.4*
Insect Growth Regulator						
Fenoxycarb	0.071	SJ-88A	44.4*	18.2	0.0	9.2
	0.140	SJ-88A	44.6*	12.6*	5.2	0.0
	0.281	SJ-87	49.5*	74.9*	8.3	0.0
	0.281	SJ-88A	25.4*	3.6	6.7	6.3
	0.281	DL-88	51.0*	7.6*	0.0	0.0
	0.281	MR-89	44.2*	25.1*	0.0	0.0
Formamidines						
Amitraz	0.140	SJ-88A	0.0	12.0	0.0	7.4
	0.140	RR-88	27.1*	5.8	0.0	6.0
	0.140	SJ-89	69.8*	13.7*	0.0	3.1
	0.140	RR-89	26.8*	59.1*	0.0	0.0
	0.203	SJ-88A	61.8*	14.5	4.7	2.9
	0.203	RR-88	36.8*	14.7*	0.0	0.0
	0.203	RR-89	58.5*	30.0*	0.0	3.1
	0.281	SJ-87	62.0*	84.9*	0.0	0.0
	0.281	SJ-88A	40.3*	36.1*	0.0	0.0
	0.281	RR-88	32.2*	36.6*	4.2	6.0
	0.281	MR-89	59.0*	17.9*	18.7	8.8
Chlordimeform	0.140	SJ-87	44.7*	19.8*	27.1	0.0
	0.140	SJ-88A	44.8*	33.4*	18.4	17.5
	0.281	SJ-87	56.4*	66.7*	6.2	6.2
	0.281	SJ-88A	37.6*	32.1*	6.4	0.0
SN 49844	0.140	SJ-89	67.6*	19.3*	16.3	10.9
	0.281	MR-89	49.8*	21.5*	6.3	7.3
	0.281	SJ-89	56.0*	22.0*	0.0	9.5
Organochlorines						
Endosulfan	0.281	MR-89	44.2*	1.0	8.3	0.0

CONTINUED,

Insecticide	Rate kg [AI]/ha	Test	% Egg Mortality 4-h	% Egg Mortality 48-h	% Larval Mortality 4-h	% Larval Mortality 48-h
Organophosphates						
Acephate	0.281	DL-88	42.7*	10.7	42.4*	2.7
	0.281	RR-89	9.9	5.8	45.8*	2.7
Azinphosmethyl	0.281	DL-88	66.2*	3.3	11.8	0.0
	0.281	RR-89	4.6	1.8	39.0	11.5
Malathion	0.281	DL-88	56.0*	5.0	0.0	2.0
Methamidophos	0.281	SJ-88A	33.8*	7.2	27.3*	5.6
	0.281	RR-89	6.9	0.0	6.3	0.0
Methyl Parathion	0.281	DL-88	45.8*	5.0	0.0	0.0
	0.281	RR-89	8.1	0.0	15.0	0.0
Profenofos	0.140	SJ-88A	47.8*	11.5	0.0	0.0
	0.140	RR-89	9.5	0.0	24.8	0.0
	0.281	SJ-88A	61.6*	10.9	8.8	9.1
	0.281	RR-88	38.2	0.0	20.2	0.0
	0.281	MR-89	81.7*	8.7	41.7*	3.1
	0.281	RR-89	59.3*	0.0	11.1	11.1
	0.563	RR-88	40.3	2.5	2.2	9.0
	1.125	SJ-87	84.9*	66.2*	24.8	0.0
Sulprofos	0.281	SJ-88A	53.9*	20.5	60.5*	11.9
	0.281	RR-89	53.0*	0.0	41.4*	0.0
Pyrethroids						
Bifenthrin	0.068	SJ-88B	43.8*	---	46.9*	---
	0.068	SJ-89	45.8*	14.4*	12.5	6.7
Cyfluthrin	0.012	SJ-88B	26.4*	---	38.5*	---
	0.028	SJ-88B	23.9*	---	68.4*	---
	0.028	SJ-89	67.8*	13.2	78.7*	9.0
Cypermethrin	0.068	SJ-87	69.5*	0.0	37.4*	41.6*
	0.068	SJ-88B	12.4*	---	44.0*	---
	0.068	MR-89	67.0*	17.9*	21.3	13.7
Esfenvalerate	0.037	SJ-88B	46.3*	---	63.6*	---
	0.037	SJ-89	56.8*	8.7	54.3*	10.7
Fenvalerate	0.140	SJ-88B	33.7*	---	32.5*	---
Fluvalinate	0.084	SJ-88B	36.9*	---	38.5*	---
L-Cyhalothrin	0.028	RR-88	34.6	17.6	51.1	5.0
	0.028	SJ-88B	54.9*	---	59.4*	---
	0.028	SJ-89	53.2*	29.4*	36.7*	21.9*
	0.034	SJ-88B	41.9*	---	50.8*	---
Permethrin	0.169	SJ-88B	47.9*	---	33.7*	---
Tralomethrin	0.020	SJ-89	45.0*	9.2	8.3	8.7
	0.021	SJ-88B	42.4*	---	45.2*	---

Treatment means are significantly higher (*) than the mean mortality observed in the control treatment in each sample prior for that respective test ($P < 0.05$, Dunnett's one tailed t-test; SAS Institute 1988).

FIG. 1. Mean mortality of tobacco budworm and bollworm eggs collected from cotton plants at different sample periods after treatment, St. Joseph, Louisiana, 1988 (SJ-88A). Insecticide treatments: Thiodicarb (0.281 kg [AI]/ha), Amitraz (0.281 kg [AI]/a), CDF (chlordimeform 0.281 kg [AI]/ha), Profenofos (0.281 kg [AI]/ha) and FENOXY (fenoxycarb, 0.281 kg [AI]/ha). Treatment mortality significantly higher than control mortality for each respective sample period at $P < 0.05$ (*) or $P < 0.01$ (**), Dunnett's one tailed t-test (SAS Institute 1988).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Thiodicarb
Amitraz
CDF
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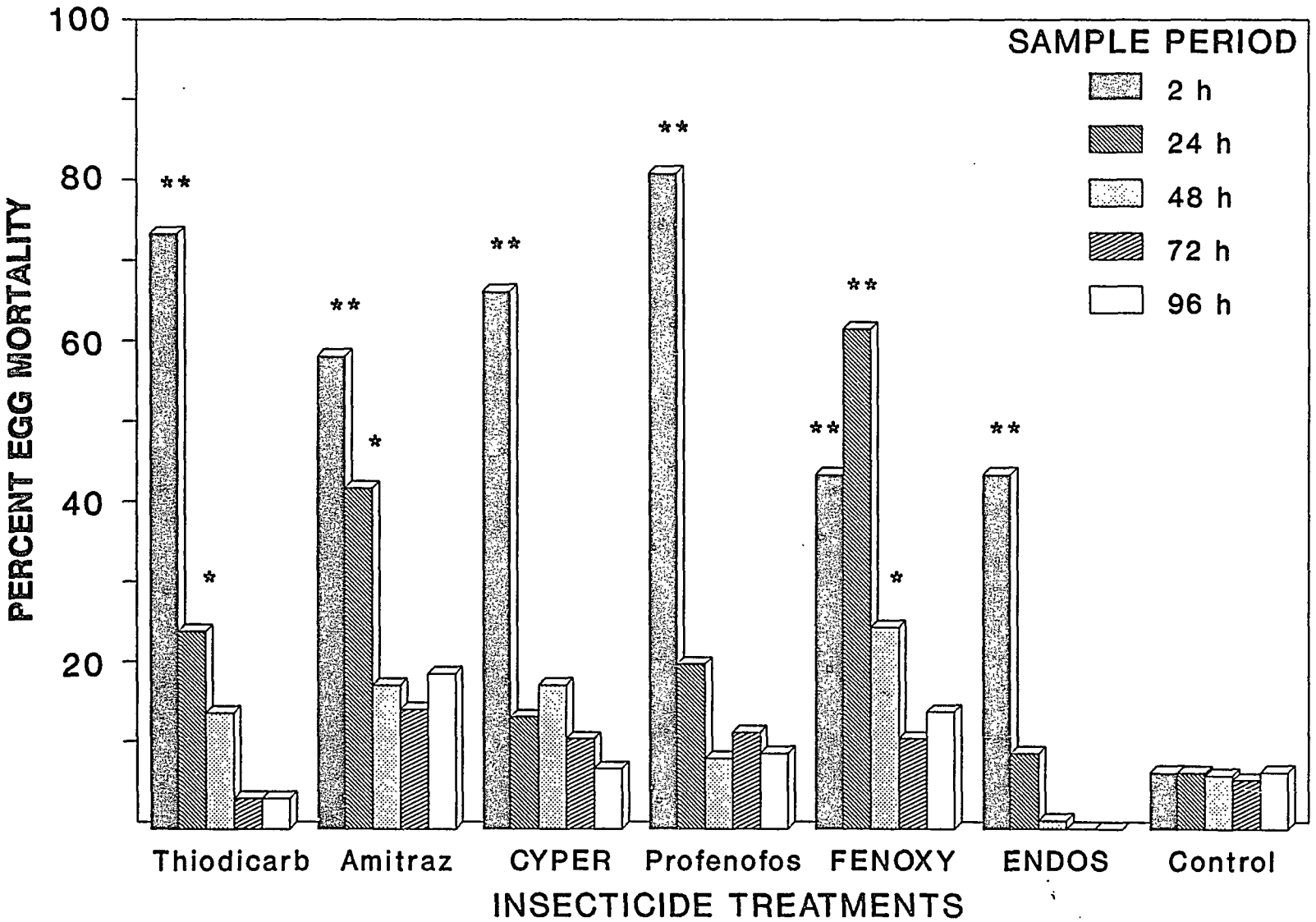
FENOXY
Control
INSECTICIDE TREATMENTS

FIG. 2. Mean mortality of tobacco budworm and bollworm eggs collected from cotton plants at different sample periods after treatment, Macon Ridge Branch, Northeast Research Station, Winnsboro, Louisiana, 1989 (MR-89). Insecticide treatments: Thiodicarb (0.281 kg [AI]/ha), Amitraz (0.281 kg [AI]/a), CYPER (cypermethrin, 0.06 kg [AI]/ha), Profenofos (0.281 kg [AI]/ha), FENOXY (fenoxycarb, 0.281 kg [AI]/ha) and ENDOS (endosulfan, 0.281 kg [AI]/ha). Treatment mortality significantly higher than control mortality for each respective sample period at $P = 0.05$ (*) or $P = 0.01$ (**), Dunnett's one tailed t-test (SAS Institute 1988).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INSECTICIDE TREATMENTS

Discussion

Egg mortality in the DL-88 trial (4 h posttreatment) was influenced by a high number of eggs (> 30% of the sample) which had been parasitized by Trichogramma spp. High egg mortality in the 4 h sample of the RR-88 probably resulted from drift of insecticide treatments to the control plots. Egg mortality in the 48 h sample of the control treatments for all tests was lower than that observed in the 4 h samples, although in the RR-89 test egg mortality remained high (28.3%) in the 48 h sample. There was no larval mortality in the 4 and 48 h posttreatment samples from the control treatments of all tests except in the SJ-88B (2.5% larval mortality) and RR-88 (5.2% larval mortality) tests.

The high level of egg mortality observed in the field trial by 4 h posttreatment is probably related to contact toxicity from direct application of the insecticide treatments to the egg. Previous studies (Wolfenbarger et al. 1974, Chalfant et al. 1979, Pitts & Pieters 1980) have attributed most of the egg mortality from insecticides as being due to direct application. Eggs oviposited after treatment may be exposed to insecticides by direct contact with residues on leaf surfaces and by vapor activity. The toxicity of insecticide vapors to insect eggs (particularly

formamidines) has been demonstrated previously (Smith & Salkeld 1966, Phillips 1971, Wolfenbarger et al. 1974).

Several studies have evaluated insecticide toxicity to tobacco budworm and bollworm eggs in laboratory and field tests and have reported results similar to those in our studies (Pitts & Pieters 1980, Gonzales & Allen 1985, Horowitz et. al. 1987, Bradley & Agnello 1988). Methomyl was one of the most toxic insecticides in laboratory tests and demonstrated high levels of initial toxicity to tobacco budworm and bollworm eggs in field tests but this activity did not persist very long in the field. Thiodicarb caused high levels of initial toxicity to eggs in field tests (51.9 - 85.7% egg mortality) and exhibited slightly improved residual activity above that of methomyl. Other studies on fenoxycarb (Masner et al. 1987) and thiodicarb (Bradley & Agnello 1987) reported egg mortality values comparable to chlordimeform 48 h after insecticide application.

The results of our field tests and that of Elzen (1989) showed the initial ovicidal activity of amitraz is comparable to that of chlordimeform. In a comparison of the toxicity of selected formamidine insecticides to tobacco budworm eggs, Gemrich et al. (1976) found no significant difference between SN 49844 and chlordimeform, but found chlordimeform to be slightly more toxic to bollworm eggs. SN 49844 demonstrated both initial and residual ovicidal

activity comparable to the activity of chlordimeform in our field tests. In other field studies, amitraz was not as effective as chlordimeform against tobacco budworm eggs (Coulon 1978, Pitts and Pieters 1980).

The organophosphates tested in the field tests generally exhibited initial ovicidal activity but did not cause significant egg mortality in the 48 h posttreatment sample, except for a very high rate of profenofos. Results of the field tests indicate that the initial ovicidal activity of profenofos was not significantly different from that of chlordimeform, but in laboratory bioassays with insecticides from several classes, profenofos exhibited the lowest ovicidal activity (B.R.L., unpublished data). Profenofos, in addition to other organophosphates, is toxic to insect eggs (Smith & Salkeld 1966, Campbell et al. 1979, Herzog & Phillips 1987, Horowitz et al. 1987), and the high level of toxicity in the present study may be related to the application rate. The organophosphate class of insecticides generally exhibits ovicidal activity comparable to that of carbamates, formamidines and organochlorines in studies that are designed to evaluate their contact toxicity to insect eggs (Smith & Salkeld 1966, Campbell et al. 1979, Herzog & Phillips 1987, Horowitz et al. 1987).

The pyrethroids also exhibited significant levels of initial ovicidal activity. The toxicity of various

pyrethroids to tobacco budworm and bollworm eggs has also been demonstrated in previous studies (Tysowsky & Gallo 1977, Pitts & Pieters 1980, Horowitz et al. 1987).

Although insecticides are absorbed by eggs at the time of treatment, they may not become lethal until further development depending on the dose that actually reaches the embryo. Noctuid eggs are very susceptible to ovicides immediately after oviposition before the membranes surrounding the embryo are fully formed (Salkeld & Potter 1953, Smith & Salkeld 1966). Insecticides may be retained in the egg until further embryonic development or the dose of insecticide becomes lethal through continuous exposure and/or metabolism. Storage either within the egg or on the chorion would increase the potency of the toxicant by increasing the overall amount received by the embryo.

Although ovicides are generally applied to target the egg stage of development for tobacco budworm and bollworm, additional toxicity to newly hatched larvae is likely to occur depending on the insecticide being used. All of the pyrethroid treatments were applied at rates recommended for control of tobacco budworm and bollworm in later larval stages and significant levels of larval mortality were observed in the 4 h posttreatment egg sample. The efficacy of pyrethroids at rates similar to those used in our tests in controlling tobacco budworm and bollworm larvae in cotton

has been demonstrated in numerous field studies (Clower et al. 1987).

The organophosphates and carbamates were used at rates (0.140 and 0.281 kg [AI]/ha) that are lower than those recommended for larval control and would not be expected to cause high levels of mortality. However, in several instances, the thiodicarb, acephate and sulprofos treatments demonstrated significant levels of toxicity to larvae. Bradley and Agnello (1988) found thiodicarb (0.140 and 0.281 kg [AI]/ha) to be highly toxic to neonate bollworm larvae during eclosion from the chorion. Tobacco budworm and bollworm larvae would be sensitive to insecticidal toxicity immediately after hatching from the egg and would require less toxicant than larvae in later developmental stages.

The residual toxicity of insecticides to newly hatched larvae is related to their persistence on the chorion and leaf surface to which the egg is attached. Larval mortality following eclosion from the egg was probably caused by chorion feeding, contact and/or oral toxicity from insecticide residues on the piece of cotton foliage upon which the egg was oviposited, or a combination of both. In the present study, less than 1% of the larvae that emerged from the egg failed to completely eclose. Gonzales and Allen (1985) and Bradley and Agnello (1988) observed considerable mortality in larvae that partially eclosed from

the egg. They concluded that this mortality was due to feeding on the chorion that had been exposed to sublethal doses of the insecticide incapable of halting embryo development.

The data presented herein suggests that the carbamate thiodicarb, and the formamidines, amitraz and SN 49844, may be alternative ovicides to chlordimeform. The persistent toxicity of insecticides in the current study suggests that ovicidal and residual larvicidal activity are two sources of mortality contributing to overall insecticide efficacy. These studies did not evaluate these insecticides with reference to the other attributes of chlordimeform (i.e. synergism of insecticides against the larval stages, modification of adult behavior, etc.). Additional information should be collected to determine if these compounds will be viable alternatives to chlordimeform.

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SUMMARY

Potential of Host Plant Resistance in Cotton To Manage Tobacco Budworm and Bollworm

Growth, development and survival of pyrethroid-resistant (PY-R) and -susceptible (PY-S and FIELD-88) tobacco budworm, Heliothis virescens (F.), and bollworm (CORN-BW), Helicoverpa zea (Boddie), were compared on four cotton lines ('Deltapine 41' [DPL 41], La. HG-660, La. HG-063 and PD-0804) and artificial diet. No significant insect and cotton line/diet interaction was observed for larval weights (5 and 9 d after initial exposure), larval stadia duration, time required for adults to eclose and cumulative mortality. The FIELD-88 and CORN-BW field strains had significantly higher 5 and 9 d larval weights compared to the PY-R and PY-S laboratory strains. No significant differences among insect strains were observed for larval stadia duration and d to adult eclosion. Larvae fed La. HG-660 and La. HG-063 cotton lines had significantly lower larval and pupal weights, required significantly longer to pupate and had higher cumulative mortality compared to those reared on flower buds (squares) of a commercial cultivar, DPL 41. The high glandulosity (HG) trait in the La. cotton lines influenced all insect strains equally, regardless of their origin and pyrethroid susceptibility.

Field trials were conducted during 1988 at the Dean

Lee Research Station and during 1989 at the Northeast Research Station to evaluate combinations of cotton cultivar/lines and insecticides against tobacco budworm and bollworm. There was no significant interaction ($P = 0.05$) between cotton cultivar/line and insecticide treatments in the 1988 experiment. Significant effects on variables were attributed to either cotton cultivar/line or insecticide treatment. Plots planted to La. HG-660 had significantly fewer larvae in plant terminals, damaged plant terminals, larval damaged squares and larval damaged bolls than were observed in the plots planted to DPL 41. Cotton cultivar/line had no significant effect on numbers on boll weevil damaged squares in this trial. The results of the 1988 trial indicated that La. HG-660 reduced tobacco budworm and bollworm infestation and damage, matured significantly earlier and produced yields comparable to DPL 41. Lint turnout and boll weights were lower in the La. HG-660 line than DPL 41. The numerical changes in the yields for the two cultivars were related to the lint turnout and boll weight of each cotton cultivar/line. Plots planted to La. HG-660 had significantly more total bolls and matured significantly earlier than was observed in the plots planted to DPL 41. However, there was no significant difference in seed cotton yields and lint yields between the La. HG-660 and DPL 41 plots. Boll weights and lint turnout were

significantly lower in the La. HG-660 plots compared to the DPL 41 plots. Cotton cultivar/line had no significant effect on fiber length or micronaire of the cotton samples. Fiber strength of La. HG-660 was significantly lower than that of DPL 41.

The insecticide treatments used in 1988, thiodicarb and lambda-cyhalothrin, significantly reduced the number of damaged plant terminals, damaged squares and damaged bolls compared to numbers in the untreated plots. These treatments also significantly increased yield above the control. Lambda-cyhalothrin significantly reduced the number of larval damaged fruit and increased yield in comparison to thiodicarb at the rates used in this test. Insecticide treatments had no significant effect on total numbers of bolls, crop maturity, lint turnout, or boll weight. However, the insecticide treated plots yielded significantly more seed cotton than was produced in the untreated plots. Insecticide treatments had no significant effect on fiber properties.

Tobacco budworm and bollworm pressure in the test plots was low during most of the 1989 season. There were no significant treatment effects on numbers of terminal larvae, larval damaged squares and percent larval damaged bolls. Significant differences among treatments were observed for numbers of larval damaged terminals and boll weevil damaged

squares. The La. FNHG-850075 plots treated with Dipel ES had significantly fewer damaged plant terminals than was observed in the DPL 41 (treated and untreated) plots. Plots of La. HG-660 (treated and untreated) and La. FNHG-850075 (treated and untreated) had significantly fewer damaged plant terminals compared to the number in the DPL 41 plots receiving no insecticide treatments. Plots planted to La. FNHG-850075 (treated and untreated) had significantly fewer boll weevil damaged squares compared to the number in the untreated DPL 41 and untreated La. HG-660 plots. No significant differences were observed among treatments in numbers of total bolls.

There were significant differences among treatments in crop maturity, seed cotton yields, lint yields, lint turnout and boll weights. The untreated plots of La. HG-660 had significantly more open bolls compared with the number of open bolls in the untreated La. FNHG-850075, La. FNHG-850075 treated with thiodicarb and La. FNHG-850075 treated with thiodicarb + Dipel ES plots. The La. FNHG-850075 plots treated with thiodicarb yielded significantly more seed cotton than the untreated DPL 41 plots, the La. HG-660 plot treated with thiodicarb and the other La. FNHG-850075 plots. The lint yield of the La. FNHG-850075 plots treated with thiodicarb yielded significantly more seed cotton than the untreated DPL 41 plots, the La. HG-660 plots treated with

thiodicarb, the La. FNHG-850075 plots treated with Dipel ES and the La. FNHG-850075 plots treated with Dipel ES + thiodicarb. Samples of cotton from the DPL 41 plots (untreated and treated) had a significantly higher lint turnout compared to the lint turnout of all other treatments in the trial. Boll weights of DPL 41 (treated and untreated) were significantly higher than boll weights of La. HG-660 (treated and untreated). The cultivar/line and insecticide treatment combinations had no significant effect on fiber properties.

Currently, pyrethroid resistance has been documented in tobacco budworm populations across the cotton belt. Alternative tobacco budworm and bollworm larvicides are more expensive and are generally not as effective as the pyrethroids against the boll weevil. Resistance management plans are being implemented in a concerted effort to maintain the efficacy of the pyrethroids, but such strategies are not likely to prevent the ultimate loss of these compounds. This study suggests the use of cotton varieties possessing tobacco budworm and bollworm resistance in combination with insecticides as an alternative scheme of management, but further research must be conducted to determine specific cotton line and insecticide treatment interactions.

Insecticide Toxicity to Eggs of Tobacco Budworm and Bollworm

A dipping technique was used to test the ovicidal activity of selected insecticides against pyrethroid-resistant (PY-R) and pyrethroid-susceptible (PY-S) laboratory strains and a field (FIELD-89) strain of tobacco budworm. LC_{50}'s for all insecticides except profenofos on eggs of the PY-R strain were significantly higher than LC_{50}'s for the same insecticide on eggs of the PY-S strain. Furthermore, all insecticides except profenofos and methomyl were significantly more toxic to eggs of the PY-S strain compared to their respective toxicity to eggs of FIELD-89 strain. Eggs of the PY-R strain exhibited resistance to esfenvalerate (13.6 times) lambda-cyhalothrin (34.9 times), and chlordimeform (5.4 times) while eggs of the FIELD-89 strain possessed resistance to lambda-cyhalothrin (11.7 times), chlordimeform (6.5 times) and SN 49844 (7.2 times). Chlordimeform and methomyl were generally the most toxic insecticides tested against tobacco budworm eggs.

In field tests conducted during 1987-1989, all insecticide treatments exhibited initial ovicidal activity (4 h posttreatment) in one or more trials. The formamidines (amitraz, chlordimeform and SN 49844) and a carbamate (thiodicarb) at 0.28 kg [AI]/ha generally exhibited residual

ovicidal activity. Residual toxicity to newly hatched tobacco budworm and bollworm larvae was generally higher for pyrethroids than for other classes of insecticides.

VITA

Billy Rogers Leonard was born in Monroe, Louisiana on November 18, 1961. He attended Tensas Academy (Louisiana Independent Private School) for his elementary, intermediate and high school education until his graduation in May of 1979. He began collegiate studies at Northeastern Louisiana University (Monroe, LA) in the Fall of 1979, transferred to Louisiana Polytechnical University (Ruston, La.) in the Fall of 1980 for two years of study and finally entered Louisiana State University (Baton Rouge, LA), where he received a B.S. degree in Agronomy (Crop Science) in May of 1984. Upon graduation he began graduate studies in the Department of Entomology (Cotton Pest Management), Louisiana State University (Baton Rouge, LA) and received a M.S. degree in December of 1987. He continued his graduate education in the Department of Entomology, Louisiana State University (Baton Rouge, LA) and is presently a candidate for the Ph.D. degree.

DOCTORAL EXAMINATION AND DISSERTATION REPORT

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Major Field: Entomology

Title of Dissertation: Management of Tobacco Budworm, Heliothis virescens (F.) and Bollworm, Helicoverpa zea (Boddie) in Cotton: Host Plant Resistance and Ovicidal Approaches

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